THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—26TH YEAR. SYDNEY, SATURDAY, JUNE 10, 1939. No. 23.

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CHRONIC INFECTION AND ATHEROSCLEROSIS.[1]

By NOBLE W. JONES and ARTHUR L. ROGERS,

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SOME years ago there came to us two patients[(1)] suffering from congestive heart failure on the basis of atherosclerosis, who influenced markedly our ideas about their underlying disease.

The first patient was a business man, fifty-nine years old, who had been suffering from heart failure for several months before entering the hospital. For another month under control we were unable to restore his compensation. As there was present a chronic and severe infection of the tonsils and both antra, we suggested to him that he permit the removal of this infection to see if it might not have some influence on the course of his heart disease. He consented readily to the proposal, as he thought he could not get well anyway, and it was done. First the tonsils were removed, and this was followed by a stormy convalescence, during which the degree of failure increased temporarily. After two weeks both antra were radically exenterated, and we were much surprised to see the patient pass through an easy convalescence, during which time the heart became rapidly compensated. The entire clinical picture changed even as one sees it change at times following the removal of a toxic goitre. In three weeks the patient was sitting up, and by the end of the month he returned home. He again took up his business and was quite well for one and a half years, when we received word that he had died suddenly from an acute coronary occlusion.

The second patient was a housewife, forty-nine years of age, who had had symptoms of congestive heart failure for two years on the basis of hypertension and atherosclerosis with auricular fibrillation, and had been confined to her bed for nearly six months. She had had a cholecystectomy performed in 1915, and in 1920 had had further attacks of pain in the right side of the upper part of the

[1] Read at the inaugural meeting of the Royal Australasian College of Physicians, December 15, 1938.

abdomen. In the hospital, under control, we were not able to compensate the patient's heart. One day during this time she had a slight chill, some right upper abdominal pain, a little fever, an increased white blood count and icterus index, and the following day there appeared slight jaundice. Because of our experience with the first patient a couple of months before, we suggested to the patient that there might be some relation between the liver infection and the condition of her heart. We asked her to consider the question of operation for the purpose of draining the common bile duct. We found the patient more willing to have the operation performed than was the surgeon to perform it. However, after all factors involved had been carefully weighed, the operation was performed, and again much to our surprise, the post-operative convalescence was easy and the heart became recompensated with surprising rapidity. In due time the patient returned home to take up her household duties again. A year later she went through a severe bronchopneumonia without decompensation of her heart, and five years later, when last heard from, she was still well. For two years the heart rhythm was normal after a course of quinidine therapy, but later the fibrillation became permanent and was controlled by digitalis.[1]

Why, one might well ask, did the dramatic relief of the congestive heart failure take place after the removal of the infection? An answer now certainly cannot be given. As time went on other patients with atherosclerotic heart disease with and without congestive failure were treated in a like manner when they had an associated chronic infection. Some were markedly benefited. Several were not.

One patient's heart condition was much improved by the removal of a severe pericemental dental infection.

Another patient, a lawyer, sixty-two years of age, who had been an invalid for six years, lived seven years in very good health after his sinus operation and then died of cardiac decompensation secondary to a severe unrelieved infective asthma of seven months' duration.

Still another patient, a business man, aged fifty-six years, suffered in 1932 from attacks of acute dyspnœa, particularly at night, and substernal oppression with distinct *angor animi*. These symptoms disappeared entirely after double radical antral exenteration and transantral ethmoidectomy. He reported a few weeks ago that he had remained well throughout these six years. However, his electrocardiograms have not changed. Negative *T* waves persist in Leads I and II, indicating some myocardial damage.

From the first patient operated upon we had only the sinus membrane for histological and bacteriological study. The arterioles lying in the depth of the membranes were noteworthy because of an almost universal subacute arteritis and a thrombotic process which occluded many of their lumina. Scattered diffusely throughout the walls of the vessels, and in the thrombi also, were many micro-organisms in the form of diplococci, as revealed by specially stained sections (Figures I and II). This picture was at marked variance with the arterioles in the sinus tissues removed from patients suffering from chronic arthritis or asthma, for instance, in which the blood vessels were slightly or not at all affected. This fact in itself was highly suggestive of a relationship to distant thromboses and led us to study further all material available. There were no tissues removed at operation from the second patient, but at later times we were able to study several gall-bladders removed from atherosclerotic patients, and in all of them there was found in the arterioles of their walls a subintimal fibrous connective tissue proliferation which caused marked narrowing of the lumen. Lymphocytic infiltration was noted particularly in the subintimal areas, but the media and adventitia were also involved in the proliferative process. Numerous diplococci were found in these proliferative areas (Figures III and IV). In both the sinus and the gall-bladder tissues the pathological process in the blood vessels was distinctly inflammatory in type.

The first opportunity to study the coronary arteries from the heart of one of our patients was in 1932. We had examined the patient, a man, aged fifty-six years, who had complained of anginal symptoms for several years, in 1930 and had suggested that there might be a relationship between a severe hyperplastic double antrum disease and his symptoms. This infection was not removed. He died suddenly in a distant city two years later from an acute coronary occlusion. The coronary arteries were considerably narrowed. Both right and left arteries were nearly occluded about one inch from their orifices, and there was a freshly organized thrombus completely occluding the circumflex branch of the left artery. The lumen of one artery studied histologically (Figures V and VI) was narrowed, owing largely to subintimal proliferation of fibrous connective tissue. Collections of lymphocytes and plasma cells were present, and also clefts where cholesterol crystals had been. Some œdema was evident in those areas where the inflammatory cells were most abundant. The outer layers of the media and inner layers of the adventitia were also involved by a fibrous connective tissue proliferation, and collections of small lymphocytes were present. Particularly in these collections of lymphocytes numerous diplococci were visible in specially stained sections. The histological picture, therefore, was that of subacute arteritis with cholesterol deposition. It presented an inflammatory character more than that of degeneration.

For comparison with the microscopic picture seen in the coronary arteries in the above case we examined the material obtained at *post mortem* examination from eleven unselected patients who died from acute coronary thrombosis. In all the same general histological picture of subacute arteritis with atheromatous degeneration was noted, and also in each one microorganisms in the form of diplococci were found in the tissues. In a few instances single and short chain cocci were seen. In these eleven cases there were associated chronic cholecystitis three times and a severe grade of pericemental infection and chronic sinus disease each twice. Up to the present time we have searched for microorganisms in the coronary tissues in fifteen cases of atheromatous heart disease and have found them without exception in each one. To be sure, the finding of microorganisms in devitalized tissues,

[1] In February, 1939, this patient returned to the hospital again suffering from cardiac failure, after having been in comparatively good health for twelve years. Her heart has again been recompensated and she has returned to her home.

and particularly in dead bodies, carries with it much doubt as to their significance; but the presence of an evident inflammatory reaction is of some value; and again, in the blood vessels of the gall-bladder and the sinus tissues removed at operation, which were living tissues, the same relation of microorganism distribution in the vessel walls was seen.

The next step in our study was the examination by the same methods of coronary arteries which did not show the presence of atherosclerotic changes. In young children, as illustrated in Figure VII, the artery walls are immaturely developed, the internal elastic membrane is very faintly discernible, and, as one would expect, microorganisms could not be found in them. Coronary arteries in the presence of essential hypertension (Figure VIII) present a picture of universal hypertrophy. The intima and media are greatly thickened and the internal elastic membrane is more prominent than in any other heart condition. There is little or no evidence of inflammation, and no deposition of cholesterol or lime salts. Many sections examined for micro-organisms failed to show them. True senile arterio-sclerosis of the coronary arteries presents a different picture from that of atherosclerosis (Figure IX). The vessels are dilated and the coats thinned out. The internal elastic membrane, however, remains distinct and unbroken. There is no fraying or fragmentation of its fibres, as seen in the proximity of an area of atherosclerosis. It would certainly seem that the explanation of Wells,[2] namely, that under the influence of stress and strain, as the years go by, the ageing of the tissue colloids which permits a loss of elasticity of the vessel wall, with a resulting thinning of it and the dilating of its lumen, must be the most logical of all explanations given. It must be remembered, however, that atherosclerosis is found associated with all other forms of heart disease. The changes due to rheumatism, to syphilis, hypertension, thyreotoxicosis and senile arteriosclerosis are usually, if not always, if sufficient time has elapsed, combined with the lesions of atherosclerosis. It is only when atherosclerosis completely dominates the clinical picture that one speaks of the atherosclerotic heart as a clinical entity.

There is much similarity in a clinical sense between the atherosclerotic and the syphilitic heart. When a wide open regurgitation of the aortic ring does not exist in the latter, the two conditions may be so similar that a differential diagnosis is not possible. Both are characterized by dilatation of the aorta and large vessels and a gross enlargement of the heart without special valve defects. Their clinical courses resemble one another also in that congestive heart failure usually begins in the fourth and fifth decades of life in each, and once it has begun recovery seldom or never takes place. The patient with either disease remains a heart invalid for some months and dies. Hence we were surprised at the clinical recovery of our patients after removal of these infections. One must not forget, too, as mentioned above, that syphilis and atherosclerosis frequently coexist in the same heart and that the therapeutic result in either may be altered by the presence of the other.

It may now be of interest to note the histological and bacteriological pictures seen in other diseases of the arteries not generally considered to be related to atherosclerosis or degenerative processes.

In *thrombo-angiitis obliterans* there is a diffuse inflammatory process involving the media, adventitia, and particularly the perivascular tissue, with a fragmentation of the internal elastic lamina and invasion of all layers of the vessel wall by polymorphonuclear leucocytes; this differs from true atherosclerosis in that there is no splitting of the internal elastic lamina. As the thrombus forms and becomes organized, new vascular channels lined with endothelium form and may simulate a thickened intima, making the vessel appear suggestive of *endarteritis obliterans*. As the process becomes chronic the leucocytes disappear and round cells are found mainly in the inner media and outer adventitia. There is no calcification at any time. Diplococci have been demonstrated in all three layers of the vessel wall, but in largest number in the adventitia and intima (Figures X and XI).

In *periarteritis nodosa* the process usually affects the medium-sized muscular type of arteries. The lesions take the form of small nodules scattered along the course of the vessel. The nodules represent inflammatory foci in which the adventitia and media are infiltrated with polymorphonuclear leucocytes and there is extensive necrosis of the media with formation of multiple aneurysms. The inflammation extends to the intima with rupture of the internal elastic membrane and destruction of the endothelium with formation of thrombi. Diplococci have been found in the media and intima, usually in association with dense collections of polymorphonuclear leucocytes and round cells (Figures XII and XIII).

In cerebral thrombosis the pathological process is largely located in an intimal thickening, in which one may see numerous cholesterol clefts, which may be present in the intracellular ground substance or even in the branched connective tissue cells. In places there may be definite atheromatous plaques. The thickening of the intima is usually not due to a proliferative process, but to a hyaline-like swelling and degeneration. The internal elastic lamina is destroyed in places; in others it may be frayed into a number of strands, which may even pass over and under the atheromatous area.

The process in the media is usually one of atrophy with a hyaline-like degeneration. In places, usually located between the intima and media, one frequently sees collections of round cells with occasional polymorphonuclear leucocytes. One also sees these areas at times between the media and the adventitia. At times these cell aggregations resemble small abscesses, and in both of these sites we have been able to identify diplococci by special

stains. The outer adventitia does not show any consistent change (Figures XIV, XV and XVI).

With the above-related data of our own studies of atherosclerosis in mind we may review briefly the present-day knowledge of this obscure problem.

Experimental cholesterol atherosclerosis in rabbits and less in guinea-pigs has been recognized by pathologists since the first work of Anitschkow[3] (1912-1930). This phase of the subject has recently been restudied in detail by Leary.[4] Bruger and Fitz[5] refer to the original observation of Murata and Katoaka (1917) that the feeding of iodine prevented the deposition of cholesterol in the arterial walls, and that thyreoidectomy abolished this protective action (Turner and Khayat, 1933). They fed pituitary gland to rabbits for the thyreotropic factor effect, and decided that when cholesterol and thyreotropic factor are given together to rabbits over a long period of time there is often a greater deposition of cholesterol in the aorta. They thought that the cholesterol content of the aorta varied directly with the average concentration of cholesterol in the blood. Rats have been quite free of spontaneous arteriosclerosis, as opposed to rabbits in particular; but more recently arteriosclerotic changes have been observed by Hueper[6] in the branches of the pulmonary arteries, near their bifurcations. Experimental arteriosclerosis thus far has not been produced in dogs, cats, foxes and monkeys. Recently, however, a hypercholesteræmia of over 600 milligrammes per 100 cubic centimetres of blood has been produced in dogs by Corwin[7] by the feeding of a fat diet and lecithin prepared from the adrenal gland. Not enough time has elapsed to indicate a relationship with atherosclerosis. Jobling and Meeker[8] attempted to produce experimental atherosclerosis in cholesterol-fed rabbits within a short space of time—thirty days—by changing the permeability of the wall of the vessel in the region of the internal elastic membrane and by increasing the permeability of the intima without other injury to the vessel. They used intravenous injection of streptococcus toxin, 2% ammonium chloride solution by stomach tube, artificial fever to 113° F., intravenous injection of peptone solution, anaphylactic shock reactions with horse serum, intravenous injection of uric acid and intravenous injection of histamine. These experiments were duplicated also in cats. All results were negative. No focal injuries to the wall and no infiltration of lipids were observed.

The experimental reproduction of human arteriosclerosis in animals has not as yet proved possible. Duff[9] recently gave a detailed and logical review of the literature and the experimental data concerning experimental cholesterol arteriosclerosis, and from an analysis of them and the known factors of human arteriosclerosis he comes to this conclusion. He believes that some form of injury to the vessel wall is the essential factor and that it antedates the deposition of cholesterol and its esters as well as lime salts. Thus far the cause or causes of injury to the arterial wall are unknown. The importance of this view, and of the observations made supporting it, lies in the significance of focal injuries by way of the *vasa vasorum*. These causes may be mechanical, chemical, infectious, but are now unknown. In a study of the aortæ from 123 persons suffering sudden death from violence, Lande and Sperry[10] concluded that there is no correlation between the concentration of cholesterol in the blood serum and the lipid content of the vessel wall. Since it has been demonstrated by Schoenheimer and by Rosenthal that there is a parallelism between the lipid content of the aorta and the degree of atherosclerosis, it follows, from Lande and Sperry's work, that there is no direct relationship between the concentration of cholesterol in blood serum and the degree of atherosclerosis present. Lande and Sperry conclude that the incidence and severity of atherosclerosis are not directly affected by the level of cholesterol in the blood serum.

That infection may play some part in the ætiology and pathogenesis of atherosclerosis has been suggested from time to time for many years. From a comprehensive study of necropsy material from the standpoint of the relation of previous infections to the presence of arteriosclerosis found at autopsy, Ophüls[11] concluded that infection of the rheumatic type played an important role. He states that the tissue changes in arteriosclerosis are partly inflammatory but to an equal extent degenerative and reparative. Benson, Smith and Semenov[12] produced by intravenous injection of streptococcus broth cultures arteriosclerotic lesions in the aortæ of rabbits on ordinary diets in 61%. In cholesterol-fed rabbits the lesions were more marked and were found in 85%. These lesions were much more extensive than those found in cholesterol-fed rabbits which did not receive bacterial injections. The arterial tissues were not examined for the presence of microorganisms.

Now that the important work of Larsell and Fenton[13][14] on the lymphatic pathways from the nose has been completed we know that there is a direct anatomical route from the paranasal sinuses into the systemic blood stream via the paratracheal lymphatic vessels to the great veins and thence into the right side of the heart and into the pulmonary bed. From this point the pulmonary tissues and other parts of the body are reached. Trypan blue injected into the paranasal sinuses of the cat passes into the chest by a lymphatic and blood stream route consisting of the sinus lymph spaces and small lymphatics, retropharyngeal and deep cervical lymph nodes, cervical lymphatics, subclavian vein, superior *vena cava*, right side of the heart and then into the pulmonary capillary bed. From here it spreads through the systemic circulation. Viable streptococci pass along the same pathway after injection into the sinuses, and particularly when injected directly into the paralaryngeal lymph nodes, which normally serve as the filters for the lymph stream from the sinus region. Cultures from the lungs, liver and spleen gave positive results within five hours of inoculation, and the authors found

streptococci histologically in the tissues even when the results of the cultures were negative. To make the evidence complete—as regards one cause, and possibly the most important cause, of atherosclerotic changes in these vessels—there remains only the necessity of repeating the work to show whether streptococci can be demonstrated both culturally and histologically in the walls of the aorta and coronary arteries, and that their mode of entrance is by way of the *vasa vasorum*.

Finally, one must mention the fundamental work of Winternitz, Thomas and Le Compte.[15] With the aid of special methods for the clearing of the tissues and the injection of the *vasa vasorum* of the artery walls these workers have demonstrated the fact that a variable pattern of vascularity exists in all three coats of the artery, and that the extent of this vascular supply of the wall depends upon the need for blood being brought to a certain point in it. The tissue changes incident to injury and repair, as seen in most tissues, are duplicated in the architecture of the artery wall. Exudation, hæmorrhage, resorption, organization, deposition of cholesterol and lime salts, and finally the establishment of new channels of a collateral circulation within the vessel's wall have been observed and evidently proved beyond question. These changes place the process of atherosclerosis in the realm of an inflammatory reaction. Slowly, step by step, the problem of the ætiology and pathogenesis of the disease seems to be clearing. Into this problem comes the most important of all forms of heart disease, and if chronic infection is finally proved to be an important factor in its causation there will enter at least a prophylactic measure for its clinical control.

In conclusion, we have pointed out that in certain instances of atherosclerotic heart disease there exists a clinical relation to infection and that the removal of the latter beneficially affects the course of the former.

The presence of microorganisms has been demonstrated in the walls of all atherosclerotic vessels in which a careful search for them has been made.

Stress is laid upon the importance of the work of Larsell and Fenton because they have demonstrated a direct pathway between the paranasal sinuses and the heart, along which streptococci have been shown to travel. And stress is also laid upon the work of Winternitz, Thomas and Le Compte because they have seemingly proved beyond question that the artery wall responds to injury in a true inflammatory manner with increased vascularity, in the same way as other tissues, and that atherosclerosis is a common result of such reactions. All of these investigations point to more than a possible relation to chronic infection.

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Legends to Illustrations.

FIGURE I.—Hyperplastic mucous membrane of the antrum, showing thrombosed arterioles. Hæmatoxylin eosin stain. Magnified × 300.

FIGURE II.—Arteriolar wall, showing bacteria stained by modified Gram stain. Magnified × 1,900.

FIGURE III.—Arteriole in gall-bladder wall in case of chronic cholecystitis associated with atheromatous heart disease. Hæmatoxylin eosin stain. Magnified × 200.

FIGURE IV.—Bacteria within the arteriolar wall from the vessel shown in Figure III. Modified Gram stain. Magnified × 1,800.

FIGURE V.—Branch of coronary artery from man, aged fifty-six years, in whom chronic hyperplastic sinusitis had long existed. Typical histological picture of atheroma, frayed and interrupted *interna elastica*. Hæmatoxylin eosin stain. Magnified × 30.

FIGURE VI.—Numerous microorganisms shown by modified Gram stain for bacteria in the wall of the artery from a man, aged forty-nine years, the subject of chronic sinusitis for many years. The black mass in the right upper corner consists of an aggregation of microorganisms. Magnified × 1,900.

FIGURE VII.—Branch of coronary artery in a child one year of age. The artery wall is immaturely developed, the internal elastic membrane very faintly discernible. Hæmatoxylin eosin stain. Magnified × 100.

FIGURE VIII.—Portion of the wall from a branch of a coronary artery in essential hypertension. There are marked thickening of the intima and media and hypertrophy of the internal elastic membrane. No evidence of inflammation or deposits of cholesterol or lime salts was evident. No microorganisms found. Giemsa stain. Magnified × 600.

FIGURE IX.—Coronary artery showing senile arteriosclerosis. There is a general thinning of the walls, but the internal elastic membrane remains intact. No microorganisms found. Hæmatoxylin eosin stain. Magnified × 60.

FIGURE X.—Branch of popliteal artery in case of *thromboangiitis obliterans*. A diffuse inflammatory process involves all coats of the artery with fragmentation of the internal elastic membrane and invasion of all layers of the vessel wall by polymorphonuclear leucocytes. Hæmatoxylin eosin stain. Magnified × 60.

FIGURE XI.—Microorganisms in the wall of the artery shown in Figure X. Modified Gram stain. Magnified × 2,000.

FIGURE XII.—*Periarteritis nodosa*. A terminal coronary artery showing an inflammatory node. Hæmatoxylin eosin stain. Magnified × 60.

FIGURE XIII.—*Periarteritis nodosa.* Coronary artery. Microorganisms demonstrated by modified Gram stain for bacteria. Magnified × 1,600.

FIGURE XIV.—Cerebral thrombosis. Shows typical histological picture of a thrombosed atherosclerotic artery. The internal elastic lamina is interrupted in places and frayed out in others. Hæmatoxylin eosin stain. Magnified × 60.

FIGURE XV.—Cerebral thrombosis, showing inflammatory response in adventitia and media. Giemsa stain. Magnified × 300.

FIGURE XVI.—Cerebral thrombosis, showing microorganisms in the wall of the artery illustrated in Figures XIV and XV. Modified Gram stain for bacteria. Magnified × 1,300.

INFECTIONS OF THE HAND.[1]

By VICTOR J. KINSELLA,
Sydney.

THE extreme importance of infections of the hand needs no stressing. Prophylaxis is the first consideration. It concerns the prevention of "hang-nails", the immediate treatment of the pricked finger and of other wounds of the hand, and the care of the hand in the simpler cases and in the earlier stages of infection.

Prophylaxis.

Rest and Elevation.

The gratifying effect of rest in helping the body to deal with wounds and with acute inflammations is well known. Rest is the first principle to be observed in the prevention of the more severe sequelæ of wounds and of minor inflammations of the hand.

The next essential principle is elevation of the injured hand above heart level. The importance of hydrostatic levels is illustrated by the difficulty in bringing about the healing of wounds of the foot and lower part of the leg in ambulant patients, as compared, for example, with wounds of the face. Symptoms are relieved and healing is accelerated when the foot is elevated. The same is true of the hand. The increased efficiency of the circulation that occurs when the hand is raised from the dependent position is of great importance. It is easy to demonstrate to patients the improvement in colour and the disappearance of venous engorgement that occur as it rises above heart level. It is important to demonstrate this to patients in order to obtain their cooperation.

It may be remarked that elevation of the hand contrasts with Bier's "passive congestion" method of treatment. This has never achieved universal favour, because its results have not compared with those originally claimed for it. It is possible that the good results observed by some have been due to rest and recumbency rather than to the elastic bandage, a possibility which appears more likely when one observes that disciples of Bier have applied the bandage so lightly that not even the superficial veins are completely compressed and no colour change appears in the limb. Moreover, the method is dangerous unless the patient is under continuous expert supervision. Its use is thus difficult in home treatment. On the other hand, the gratifying and immediate results of rest and elevation are beyond question and can easily be secured in the home.

To be effective these principles must be thorough, and they cannot be thoroughly applied while the patient is going about his daily task. In the less severe cases he should be reclining in an easy chair, with the affected hand resting and elevated above heart level by pillows on a table alongside the chair. No doubt many of these patients recover, even if they are allowed to work; but in these cases it is better to err on the side of excessive care, for it is not always possible to tell whether an infection is going to be minor or major, and whether an injury is going to heal by first intention or become infected. A little "fussiness" in the early stages will save many hands, and a few days' rest at the beginning may save weeks or months later. In more severe cases the patient should be in bed, the pillows being placed longitudinally in the bed alongside the patient, or perhaps on a bedside table.

In minor infections affecting the fingers a voluminous dressing and a bandage secure the necessary immobility. Otherwise, a splint should be incorporated in the dressing, to hold the hand in the position of rest—wrist dorsiflexed, fingers and thumb in the position of holding a small tumbler.

The carrying out of these measures is more successful in private practice than in public hospital work. A much larger proportion of the patients with minor injuries and infections of the hand, who are at present given advice in the casualty department and sent home, should be admitted to hospital. Of course the difficulty is bed accommodation; but in some cases at least bed space would be saved by the prevention of the more serious type of hand infection.

The Prevention of "Hang-Nails".

"Hang-nails" are a source of digital infections and can easily be prevented (Figure I). They are

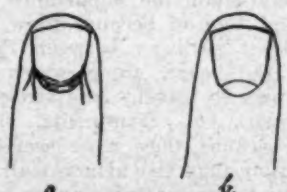


FIGURE I.

a: The unmanicured nail, showing the eponychium adherent to the nail and lines of skin cleavage with developing hang-nails. b: The manicured nail.

brought about by adhesion of the edge of the eponychium to the nail. As the nail grows, it drags with it this edge of skin, the cuticle of which becomes stretched out over the nail, and the skin is thus split along the lines of cleavage which can

[1] Based on lectures delivered in the 1935 and 1938 revision courses of the New South Wales Permanent Post-Graduate Committee in Medicine.

be seen converging on the margin of the nail fold. Prevention lies in keeping the eponychium free of adhesions to the nail, and pushed back so that the "lunule" or crescent of clear nail is visible at the base. This manicuring must be correctly done, because rough unskilled treatment of the nail fold is in itself an important cause of infection. It will usually be found impossible to carry out this procedure when the skin is dry. It is usually necessary to soak the hand for at least ten minutes in hot, very soapy water. When this has been done the adherent pieces of tough cuticle (epidermis) which bind the eponychium to the nail surface will be found softened so that they can easily be rubbed away and the eponychium pushed back by a suitably shaped blunt instrument, preferably the "orange stick" sold for the purpose by the pharmacist. When this procedure has been carried out after a period of neglect, a feeling of comfort about the nail fold is noticed, which throws into relief the barely noticed slight discomfort previously caused by the pathological stresses in the perionychium.

General Principles in Operations on the Hand.

Light ether anæsthesia is best.

Ethyl chloride is contraindicated, because it is more toxic and it makes the surgeon hurry. Local anæsthesia is contraindicated in infectious because it is ineffective and cramps the surgeon's work; moreover, it often leads to a flare-up of infection. Many cases have occurred, and some have been reported (G. Lambert and J. Syners, *"Gangrène des doigts consécutive à l'anesthésie loco-régionale", Revue de chirurgie*, Volume LII, December, 1933, page 741) in which local anæsthesia has caused severe tissue damage and gangrene of the fingers, even in the absence of infection.

A tourniquet should be used. This should not be of rubber tubing, on account of the danger of tourniquet paralysis. The best tourniquets are the cuff of the sphygmomanometer, the inner tube of a bicycle tire, or a rubber bandage which may be cut from a disused inner tube of a motor tire.

A firm support for the hand is a necessity. It is very dangerous to use a scalpel on a hand which is rolling about uncontrolled. Therefore a gloved assistant should hold the hand in position upon a side table.

The casualty department does not offer the requisite facilities for operations on the hand, and in all but the very simplest cases the patient should be taken to the general operating theatre.

The importance of a good knowledge of the anatomy of the hand is self-evident. A systematic account of this would be out of place in this paper, but some points of first importance are mentioned in the text and are made clear in the diagrams.

Iodine is an excellent antiseptic for the clean unbroken skin. It penetrates the superficial layers of the epidermis, which must be dry before the iodine is applied. But it is inactivated by blood and serum, and, like most effective antiseptics, it damages subcuticular tissues. It should therefore not be used in the wound. Acriflavine, on the other hand, is effective and comparatively non-toxic. A contaminated wound should be packed with gauze soaked in acriflavine during the cleansing of the skin; but this use of acriflavine must not be considered a substitute for the subsequent thorough *débridement*.

Swelling on the dorsum of the hand is usually due to œdema, but it may be due to pus, especially when osteomyelitis is present.

Avoid "wicks". They have no capillary action on pus, and should really be called "plugs" or "bungs". In some incisions a piece of "Vaseline" gauze is sometimes permissible for the first twenty-four hours, to prevent the lips of the incision from becoming stuck.

The hand must be splinted in the position of rest before the patient is sent back to the ward.

The Immediate Treatment of the Wounded Hand.

A careful examination should be made, with special attention to the possibility of injury to nerves and tendons. An X ray examination is necessary in some cases.

The patient should be put in hospital and repair of the wound undertaken in the operating theatre, with provision for a general anæsthetic. A gloved assistant should be present. A tourniquet should be applied. The skin should then be painted with iodine; if it is very dirty it should first be cleansed with ether soap and water and then dehydrated with spirit. Gaping wounds should be packed before this procedure with ribbon gauze soaked in a one in one thousand aqueous solution of acriflavine. The operator holds the limb during the cleansing by throwing a sterile towel over it. After the sterile towels have been draped over the limb, gauze packing is removed from the wound and damaged and dirty tissue is excised (*débridement*). Attention is given to damaged nerves and tendons. The wound is closed by interrupted sutures. If bruising or contamination has been severe the wound is loosely closed and a "cigarette" (rolled glove rubber) drain may be used for twenty-four hours. Splinting is provided, appropriate to the particular injury. The patient is sent back to bed, and rest and elevation are secured by an appropriate arrangement of pillows.

The Immediate Treatment of the Pricked Finger.

The immediate treatment of the pricked finger is of special importance to the surgeon or pathologist whose finger may be pricked by a contaminated needle. The following procedure is advisable:

1. If the epithelium is thick, its surface layers are shaved off with a razor, in order to facilitate an attack on the organisms introduced into the skin.
2. The area is painted with iodine.
3. The finger is made to bleed by squeezing the base.
4. Rest and elevation are then secured as previously described.

5. Water and moist dressings are avoided.

6. Cultures should be made from the contaminated needle, so that if the first signs of inflammation appear the appropriate medical treatment may be vigorously commenced.

7. When it is known that the needle or knife blade has become contaminated by virulent organisms, for example during the course of an operation upon grossly infected tissues, it would be advisable to introduce into the wound a hypodermic needle and inject under pressure, so as to infiltrate the tissues, a one in five hundred solution of acriflavine. I have not yet had the opportunity of employing this method. It is advocated by L. P. Garrod and G. L. Keynes in an article which repays study ("Surgical Procedures in General Practice: The Use and Abuse of Antiseptics", *The British Medical Journal*, Volume II, December 18, 1937, page 1286), and from which I have taken the following excerpt:

> Here is a small wound which is known to contain dangerous bacteria. They are in the cavity of the wound, and have not yet invaded the surrounding tissues. Can they be destroyed, and if so, how? This question has been answered by abundant experiments in which both incised and punctured wounds in mice have been infected with virulent streptococci and treated by subsequent application or injection of antiseptic solutions. Such substances as phenol and mercury perchloride are powerless to prevent the spread of infection, but compounds of the diamino-acridine series will regularly achieve this. They owe this power to an enormous capacity for restraining the growth of *Streptococcus pyogenes*, effective in dilutions of 1 in 100,000 or even higher, and to the fact that this capacity is unimpaired by the presence of serum or blood. They are also comparatively non-toxic; leucocytes retain their motility in a 1 in 1,000 solution of acriflavine in spite of being deeply stained by it. No other class of antiseptic has these properties or even approaches them. The indications in these circumstances are therefore clear. A dressing saturated in 0·2% acriflavine should be applied to the superficial scratch or cut and retained for two hours.

Later Treatment of the Wounded Hand.

When the patient is first seen some hours after infection has been established in the wound, and if the exact nature of the infection is as yet in doubt, the following treatment is advisable. The patient should be put to bed, with pillows under the arm. The surface epithelium should be shaved and painted with iodine. A hygroscopic dressing, such as magnesium sulphate and glycerine paste spread upon lint, should be applied. This should be made softer than the Australian Pharmaceutical Formulary preparation and the phenol should be omitted. This dressing would probably be sufficient splint for the finger. When the damage is more widespread a splint should be used, extending from the elbow to the finger tips, and shaped to keep the hand in the position of rest. Aluminium or a plaster slab is convenient.

Sulphanilamide should be given. An initial large dose of three grammes (or six tablets) is suitable for the average adult, and then after eight hours two tablets should be given every four hours. The dose should, of course, be reduced and the drug finally suspended, as soon as the clinical condition warrants. Probably the use of the drug for one week would be sufficient, the large dose being maintained for the first two or three days. It has been shown that it is rapidly absorbed and rapidly excreted, except in the presence of renal inefficiency, and four-hourly doses are necessary if a high blood concentration is to be maintained. I have used, with satisfaction, "Proseptasine", which is stated to be less toxic than other members of this group of compounds. Glauber's salt and Epsom salt are forbidden. Moderate blueness of the patient need not cause alarm nor lead to immediate suspension of the drug.

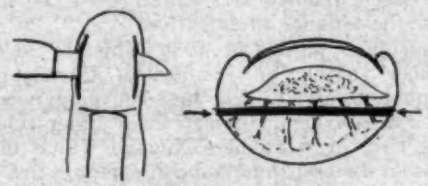
Lymphangitis.

If lymphangitis develops the signs are diffuse, and the typical red lines and lymphadenitis may appear.

The patient should be put to bed and the hand elevated. Hot foments should be applied to the whole arm, and hot-water bags should be used. "Proseptasine" should be given, and if toxæmia is pronounced, streptococcal antiserum. Operative interference is contraindicated, except for the evacuation of small abscesses which may form in the later stages.

Localized Subcutaneous Collections of Pus.

If localized subcutaneous collections of pus form, for example felons or pulp infection, the signs are localized. Hardness develops and there is no fluctuation. The patient should be put to bed and the hand elevated. Operation under general anæsthesia should be performed; for the types of incision see Figures II and III. It is important to note that



(a). (b).

FIGURE II.

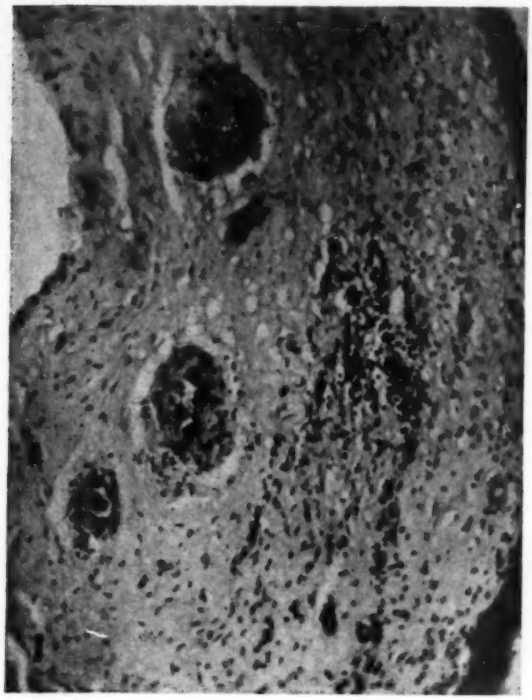
a: Method of opening a felon, showing the relationship of the distal flexion crease to the distal end of the tendon sheath. *b*: Transverse section of the terminal phalanx, showing the relationship of the incision to the subcutaneous fibrous bands.

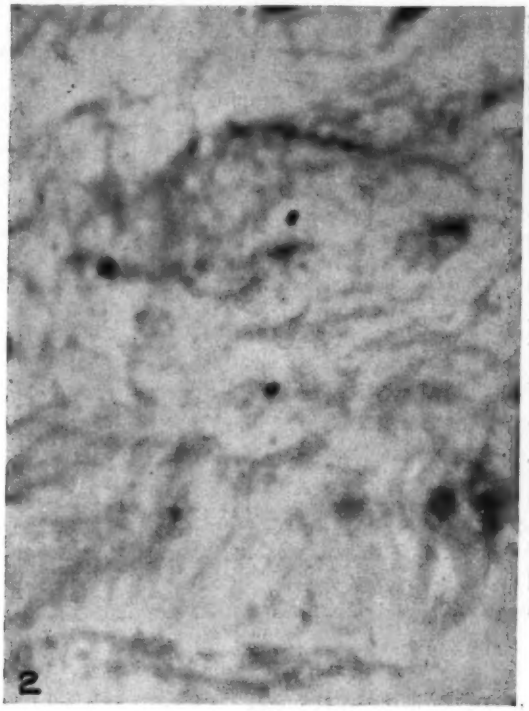
when the middle and proximal phalanges are involved the tendon sheath is in grave and imminent danger, and tenderness over the sheath in the palm may indicate a serous effusion into the sheath, which clears up when the pus is freely drained. General anæsthesia should be dispensed with only when the pus is subcuticular.

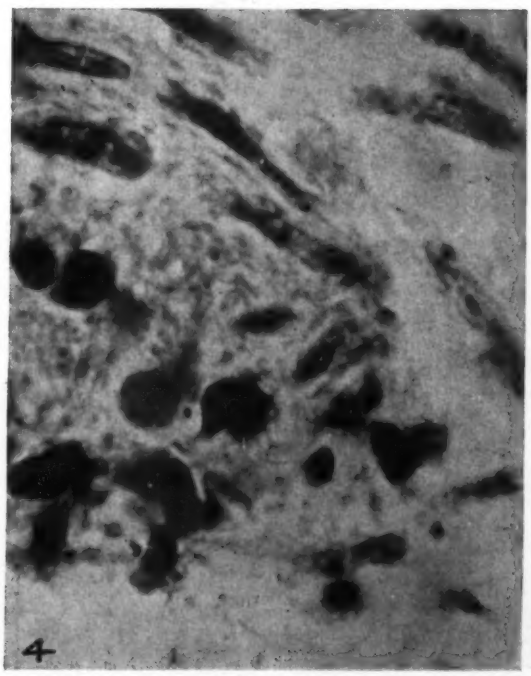
Acute Suppurative Teno-Synovitis.

The signs of acute suppurative teno-synovitis are the following: (i) the finger is held in flexion; (ii) the finger is symmetrically enlarged; (iii) the sheath is tender; (iv) pain and resistance occur on passive

ILLUSTRATIONS TO THE ARTICLE BY DR. NOBLE W. JONES AND DR. ARTHUR L. ROGERS.







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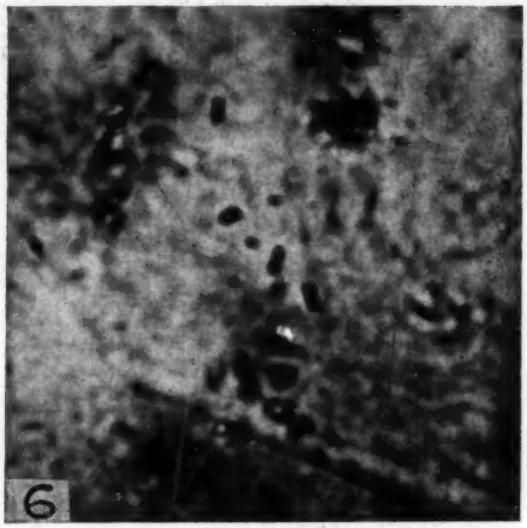

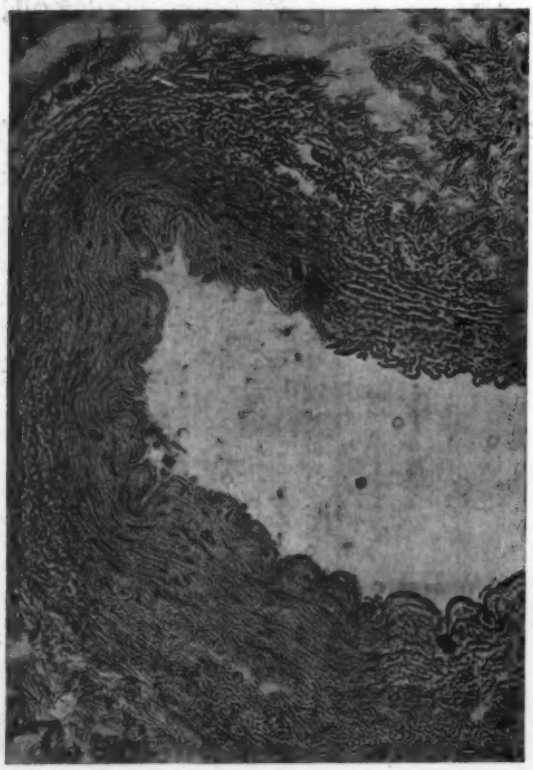

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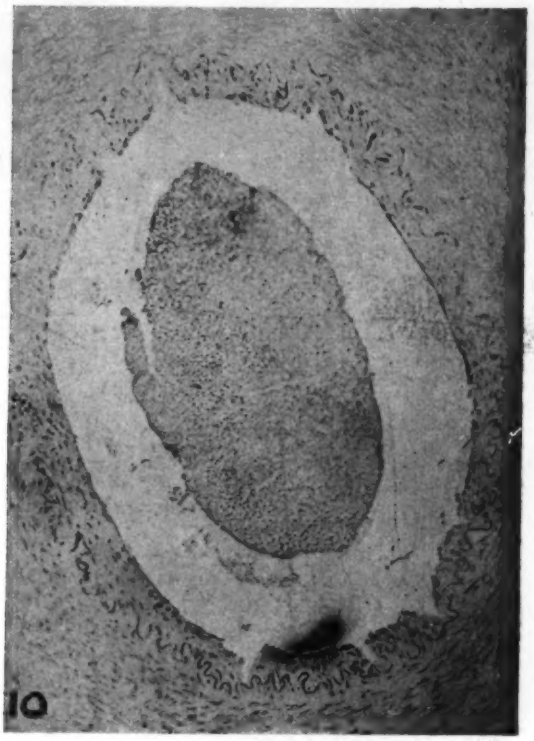

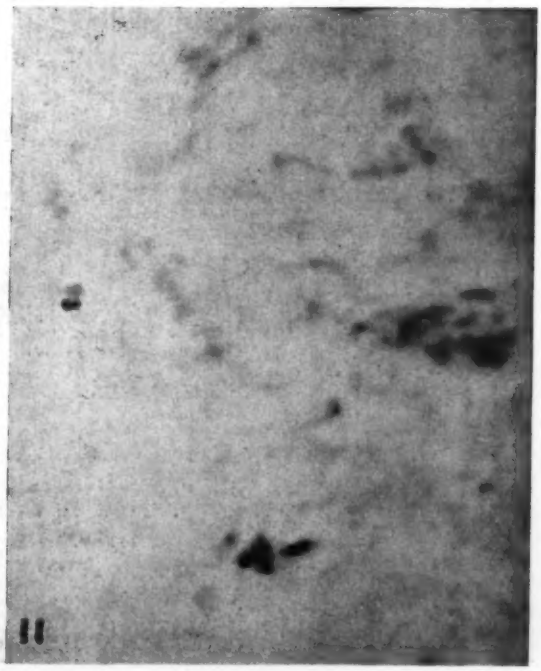

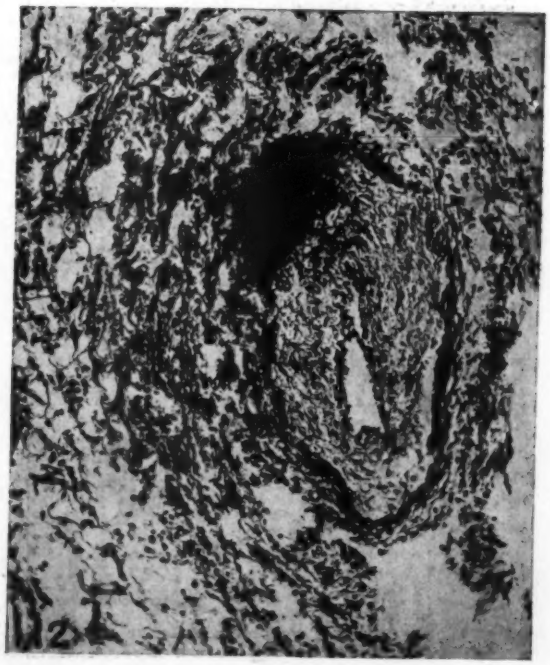

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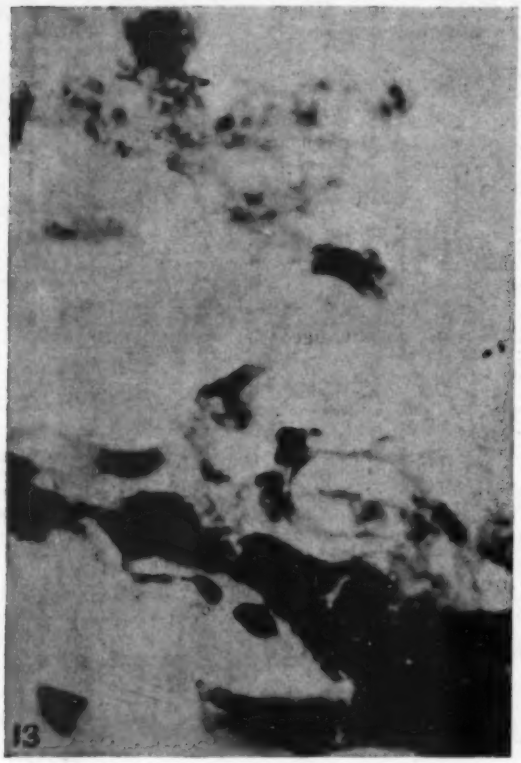

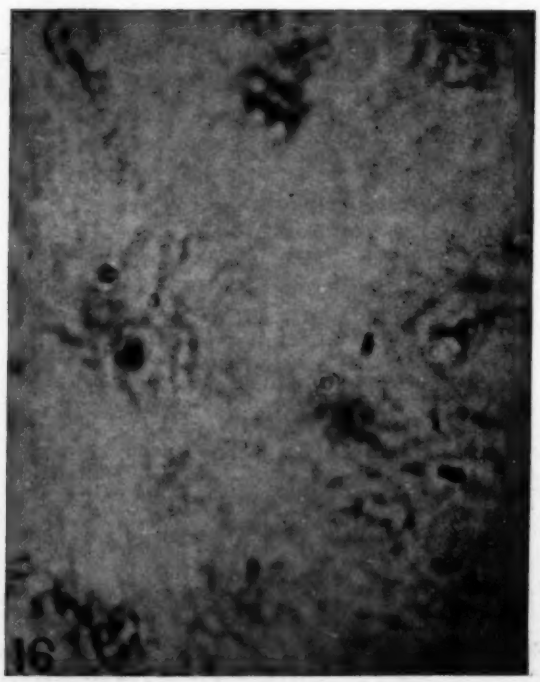

extension of the finger; (v) the exact site of the primary injury indicates the likelihood or otherwise of sheath infection. It should be remembered that the synovial tendon sheath ends distally at the distal digital flexion crease.

The first dose of "Proseptasine" (six tablets) should be given while preparations for operation

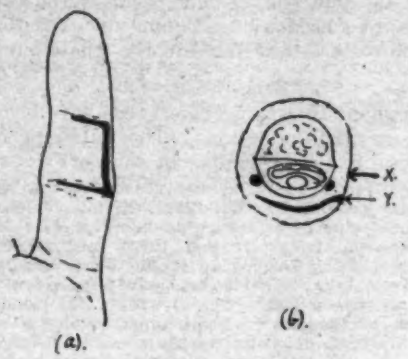

FIGURE III.

a: Showing how a subcutaneous collection of pus is drained over the middle or proximal phalanx. An L-shaped incision raises a triangular flap, or a third incision makes a quadrilateral flap. Flexion creases are not crossed. *b*: Transverse section of the middle or proximal phalanx, showing the relationship of the flap *Y* to the digital nerves and tendon sheath.

are being made. Early incision is necessary (see Figures III, *b*, and IV). The tendons slough in twenty-four to forty-eight hours if the pus is not

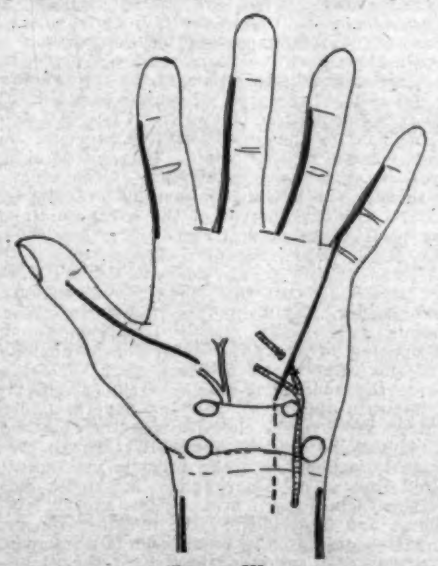

FIGURE IV.

Incisions for tendon sheath infections.

evacuated. Drainage is not used in the fingers. The wound is dressed with "Vaseline" gauze. In the palm or forearm drainage by rubber dam or "Vaseline" gauze is used for twenty-four hours. A dorsal splint is put on before the patient is returned to the ward. This is made of plaster or of aluminium, extends from the elbow to the finger tips, and is so bent that it holds the wrist in moderate dorsiflexion and the thumb abducted, and allows the metacarpo-phalangeal and interphalangeal joints to rest in moderate flexion.

The patient is kept in bed and the hand is elevated. Hot foments are applied every three hours for twenty-four to forty-eight hours. Foments are preferable to the arm bath, because the latter does not remain sterile and is more likely than foments to encourage a mixed infection. Moreover, it is not practicable to maintain the desirable degree of elevation when the arm is in a bath. After the first twenty-four to forty-eight hours "Vaseline" gauze is used and changed once a day. Active movements are encouraged after the first twenty-four to forty-eight hours if signs of toxæmia have disappeared and local signs have subsided. These may be carried out with advantage in a hot air bath. Passive movements may be of value. Kanavel advocates them and Böhler deprecates them.

FIGURE V.

Incisions for opening of the mid-palmar space.

Fascial-Space Infection.

The two deep fascial spaces (deep palmar and thenar) are separated along a line corresponding with the shaft of the third metacarpal.

The signs of fascial-space infection are the following: (i) flattening or convexity of the palmar concavity, or ballooning of the thenar eminence; (ii) tenderness over the area concerned; (iii) induration in the thenar eminence if pus is present in the thenar space (induration is not a helpful sign in the mid-palmar space); (iv) the site of the primary infection—the loose areolar tissue around the lumbrical muscles provides a ready channel of spread from fingers to palm.

Operation should be undertaken; the incisions are shown in Figures V and VI. A "cigarette" drain is left in for twenty-four hours, and the hand is

splinted. The patient is kept in bed, the hand being elevated. Hot foments are applied every three hours for one or two days, and then "Vaseline" gauze dressings are used. Active movements are begun after one or two days.

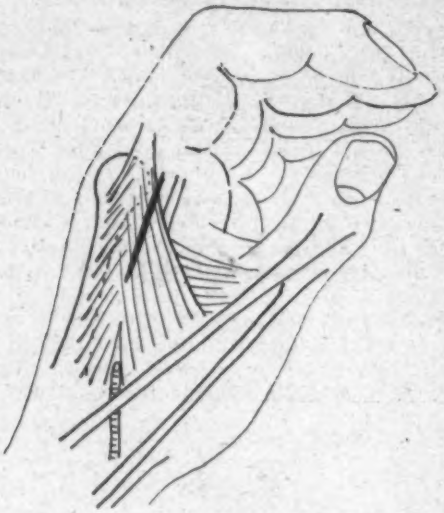

FIGURE VIa.
Incision for drainage of the thenar space, as seen from the lateral aspect of the hand.

Paronychia or "Run-Round".

Paronychia is due chiefly to hang-nails. It may disappear with conservative treatment—rest, elevation and the application of hot foments. If pus forms, it may sometimes be evacuated if the eponychium is gently pushed back. If the pus has got under the edge of the nail it will be necessary to remove that edge. This can be done in most cases almost painlessly; the eponychium is pushed back

FIGURE VIb.
The incision as seen from the dorsum.

and the nail edge is cut off with a stout pair of curved pointed nail scissors. In some cases, when the "run-round" is more extensive or when the pus extends more proximally under the nail, it may be necessary to make an incision and turn back a triangular flap of eponychium before the little abscess can be effectively unroofed.

Osteomyelitis and Suppurative Arthritis.

In the presence of osteomyelitis and suppurative arthritis amputation should be performed. This should be delayed until the original acuteness of the inflammation has settled down, by which time there will be no danger of a post-operative exacerbation. The delay should not be too great, because the pus-riddled part becomes the centre of a widespread chronic inflammatory reaction and causes fibrosis and stiffness in the whole hand.

Surgical Anatomy.

Throughout this paper references have been made to the several illustrations. The following descriptions amplify the legends.

Figure II, a: The distal end of the tendon sheath is accurately marked by the distal digital flexion crease. When the transfixion incision is being made this crease must be avoided by at least three millimetres. The sharp edge of the knife-blade should be directed distally. It is advisable to leave a bridge of undivided skin at the tip of the finger. This prevents the tendency to retraction of the flap in healing and avoids a sensitive scar in the most important tactile area.

Figure III, b: The important volar digital nerves are shown lying antero-laterally on the tendon sheath. These have the importance of a special sense organ and should be avoided when possible. The incision for the opening of the tendon sheath should fall behind these, in the direction of the arrow marked *X*; that is, it should be mid-lateral. This mid-lateral incision has the further advantage of avoiding bow-string prolapse of the tendons and a scar on the anterior or bearing surface of the sheath. The undercutting incision for a volar flap should pass in front of the nerve. Great care is needed in making this incision.

Figure IV: The incisions for the opening of the synovial sheaths are shown. They are limited distally by the distal flexion creases. Before the radial and ulnar bursæ are opened it is most important for the surgeon to feel deeply with the tip of the thumb and recognize the four bony points that mark the pillars of the transverse carpal arch or ligament. The bursæ, of course, pass beneath this arch. If it is not accurately localized there is a tendency for the incision for the ulnar bursa to be made too near the ulnar margin of the palm. This mistake may be disastrous, because the deep division of the ulnar nerve plunges through the hypothenar muscles and passes round the medial (or ulnar) border of the bursa on its way to the deep part of the palm. For this reason the advice given by N. C. Lake ("Surgical Procedures in General Practice", *The British Medical Journal*, Volume II, October 15, 1938, page 798), that this bursa should be opened on its ulnar border, should not be followed. When the bony points have been properly localized the incision for the opening of the bursa is found to approach the centre of the palm in its proximal part. This incision will divide the superficial palmar arch (which must be divided between ligatures) and the superficial branches of the ulnar nerve to the cleft between the ring and little fingers. It may be carried proximally through the transverse carpal ligament into the forearm, as indicated by the dotted line. The ulnar artery is shown; its distal branch (superficial palmar arch) passes superficialy to the ulnar bursa and is divided by the incision; the proximal branch passes deep to the bursa and is accompanied by the important deep muscular branch of the ulnar nerve. It is best first to make the opening in the first infected part of these bursæ (usually the distal or digital part) and then to dry the incision. The proximal (palmar or forearm) part of the bursa is then pressed upon. If pus appears in the incision it can be taken that the incision should be prolonged proximally. As a guide it is a useful manœuvre to pass a probe proximally up the sheath and then cut down on the probe, having regard to the anatomical points here discussed.

The incision for opening of the radial bursa must at all costs avoid the branch of the median nerve which supplies the thenar muscles, as preservation of this nerve may make the difference between a useful and a useless hand. The nerve in question crosses the bursa about a thumb's breadth distal to the distal border of the transverse carpal ligament (marked by the bony points).

The proximal extension of the radial bursa should not be approached from the front because the median nerve lies in front of it; the incision advocated in the above-mentioned article appearing in *The British Medical Journal* should not be used. The bursa should be approached by an incision near the radial margin of the forearm, just as the proximal border of the ulnar bursa can be opened along the ulnar border of the forearm. In order that these lateral approaches should be safe, the scalpel should divide only the skin and fascia, the bursa being opened by a closed artery forceps, which is pushed into it and then opened when pus appears.

Figure V: The incision for opening of the mid-palmar space is shown. It divides the web between the fingers, on both volar and dorsal aspects, and passes up the palm for no more than 1·5 centimetres. An attempt is thus made to avoid the digital nerves in the cleft. Closed artery forceps are then passed proximally (as indicated by the dotted line) in a plane behind the flexor tendons and in front of the metacarpals with their interossei. Care is taken that the forceps do not overstep the line of the middle metacarpal.

Figure VIA: The precise anatomical points governing the approach to the thenar space are shown. The incision is made parallel to the distal end of the second metacarpal and on a plane slightly anterior to it, and is limited proximally by the necessity for avoidance of the radial artery, as this vessel passes between the two heads of the first dorsal interosseus muscle. The distal fibres of the first dorsal interosseus muscle are incised. The forceps are then pushed proximally and slightly forwards into the thenar space, which lies in front of the *adductor pollicis* muscle and is shown projecting beyond the dorsal border of the interosseus muscle and behind the tendons to the index finger. The space wraps around the distal border of the *adductor pollicis* onto its dorsal surface, where it intervenes between the *adductor pollicis* and the first dorsal interosseus muscle. The forceps should not be pushed medially beyond the third metacarpal.

Figure VIB: The incision lies on the ulnar side of and parallel to the important skin crease which provides free mobility for the thumb. This skin crease must be respected by the surgeon.

CROSS-INFECTION BY THE CORYNEBACTERIUM DIPHTHERIÆ IN DIPHTHERIA WARDS.[1]

By HAROLD WILSON,
From the Kolling Institute of Medical Research, Royal North Shore Hospital, Sydney.

RECENT work has shown that it is possible to demonstrate that cross-infection by *Corynebacterium diphtheriæ* is frequent among patients in diphtheria wards. These observations have an important bearing on hospital administration.

Glass and Wright (1938) accepted the simultaneous or consecutive isolation of more than one bacterial type from the same patient as evidence that the *Corynebacterium diphtheriæ* was transferred between patients (cross-infection). This had been reported previously by Robinson (1934), by Cooper *et alii* (1936) and by Kemkes and Steigler (1937).

Of patients in hospital for a period of one month, Glass and Wright found cross-infection in 19·6%; from one to two months, in 28%; from two to three months, in 68·3%. Change in virulence, or the finding of *Corynebacterium diphtheriæ* on the throat swabs of patients who previously had not harboured this organism, was accepted also as evidence making for the same conclusion. The finding of *Corynebacterium diphtheriæ* on swabs from patients who were previously "negative" may be received as adequate evidence of cross-infection, with the limitation that occasionally *Corynebacterium diphtheriæ* is not found on the first or on every swab of a diphtheritic lesion from which it is eventually isolated in numbers. They observed, further, that only about one-fourth of patients are kept in hospital for more than two months for purely clinical reasons; the remaining three-fourths are kept because of the repeated isolation of *Corynebacterium diphtheriæ* from swabs. Cross-infection was found in 43 out of 51 of these patients (82%).

Cross-infection among patients with diphtheria is therefore apparently responsible for a heavy unnecessary cost to hospitals that receive these patients into wards in which they are allowed to mix with one another. The risk to the patient is not great and there are usually no clinical signs of reinfection. Since most of the patients are children, the economic loss to them through their being kept in hospital for an inordinately long time is not urgent enough to compel attention, although the loss of school time is often considerable.

The opinions of various authorities on accidental infection in wards have been summarized recently in this journal (Sinn, 1939). Examples were given of accidental infection by the microorganisms of most of the common infectious diseases. The present report is concerned with some experiences with diphtheria only.

During the past year in the Royal North Shore Hospital diphtheria has been generally mild. It is the practice when the patient is clinically ready to be discharged, to take a swabbing of both the throat and the nose on alternate days; the patient is discharged when no *Corynebacterium diphtheriæ* has been isolated from three consecutive pairs of swabs. The clinical state of few patients has compelled them to remain in hospital more than a month. Patients in hospital longer than this were usually there because *Corynebacterium diphtheriæ* was repeatedly isolated from swabs. The average number of days spent in this hospital last year by 111 patients with very mild faucial diphtheria was 44·9. It is important to determine, therefore, if the length of stay in hospital would be shortened by the prevention of cross-infection.

Although the number of patients investigated was, for reasons given below, too few to determine this question, this report is presented in order to

[1] This work was carried out with the aid of a grant from the Kolling Institute of Medical Research, Royal North Shore Hospital of Sydney, together with assistance from the Commonwealth Government and the National Health and Medical Research Council.

emphasize the occurrence of cross-infection in diphtheria wards and to draw attention to some of its implications.

Materials and Methods Used in the Investigation.

A swab of the nose and throat of every patient and nurse in the isolation block was taken each week for six weeks, except in the fifth week; local applications were withheld during the week-end, and the swabbing was done first thing on Monday morning.

The bacteriological technique of Anderson, Goldsworthy and Ward (1936), as modified by Wilson and Goldsworthy (1938), was used, a Löffler slope and the half of one plate of Clauberg's (1931) medium being inoculated from the throat or nose swab of each patient. Each strain was isolated in pure culture and examined in glucose, saccharose, starch and glycogen, and on sheep or guinea-pig blood-agar plates (Wilson and Goldsworthy, 1939).

The isolation block is a crazy compound of cottages, comprising one ward of 16 cots and 12 rooms each of from one to three beds or cots. An attempt was made to isolate patients singly or in groups of two or three for the whole of their stay in hospital, but lack of accommodation made this impossible. It was easy to observe that patients mixed; but owing to the unsuitable accommodation it was practically impossible to be sure they had not mixed. It was possible finally to divide only 48 patients, from whom 368 swabs had been taken, into two groups. Group A comprised 33 individuals, children and adults, who were isolated, either singly or in separate groups of two or three. No individual or group of individuals, however, was strictly isolated during the whole time; indeed, although fresh contacts were limited as much as possible, new patients had to be introduced into some of the rooms which were already occupied, and some of the old patients had to be moved from room to room. But there was no free mixing among the individuals in group A such as occurred in group B, which comprised 15 children mixing freely in one common ward.

Individuals infected by types *gravis* and *mitis* were distributed as equally as practicable through both groups, which comprised patients in whom the disease was mild or but moderately severe.

It is the practice for the nurses to wear gowns; masks are worn when an unusually sick patient is nursed, but otherwise they are not generally worn.

Results.

In Table I are shown the frequency of occurrence of *Corynebacterium diphtheriæ* and the types isolated from swabs of the individuals of groups A and B.

No individual of group A, but five of group B, showed evidence of cross-infection; from three of these five two types of *Corynebacterium diphtheriæ* were isolated from different swabs; from two individuals two types were isolated from one and the same swab.

From 17 nurses 180 swabs were examined. From one nurse, who was on night duty, *Corynebacterium diphtheriæ* was isolated from five of seven throat swabs. From four swabs it was of type *mitis* and was avirulent; from one swab it was of type *gravis* and was virulent.

Discussion.

It has never been shown that change of the type of *Corynebacterium diphtheriæ* is due to variation or that there is any genetic relationship between strains of the several types. There seems no reason to qualify the opinion of Robinson (1934) and of Cooper *et alii* (1936), with which Glass and Wright (1938) concur, that the balance of evidence seems to indicate that the three types of *Corynebacterium diphtheriæ* are stable.

Because of the unsuitable accommodation in the isolation block it was impossible to control completely intercourse between patients; this was particularly true of children. The results of much work had to be rejected because of doubt as to whether the patients had mixed; the results given here are consequently too few to be statistically significant. Cross-infection was manifested in about 10% of all patients.

Since the methods used were capable only of distinguishing between types of *Corynebacterium diphtheriæ*, but were incapable of distinguishing between different strains of the same type, it is probable that cross-infection really occurred more often than was detected. Also the disease was mild in all patients, none of whom had nasal discharge; if the disease had been severe very many more bacilli would have been discharged from the nose and mouth. The material was therefore not the most favourable for proving cross-infection.

When the two groups were compared (see Table I), cross-infection was observed in one-third of the individuals who mixed in a common ward (group B), but in none of those who were relatively isolated (group A).

TABLE I.

The Incidence of Corynebacterium diphtheriæ, and the Types Isolated from Swabs of the Individuals of Groups A and B.

Group.	Number of Individuals Examined.	Number Harbouring *Corynebacterium diphtheriæ*.	Number in whose Swabs *Corynebacterium diphtheriæ* was not Found.	Bacterial Type.			Mixed Types or Change of Type.
				Gravis.	*Intermedius.*	*Mitis.*	
A[1]	33	22	11	15	1	6	0
B[2]	15	15	0	11	0	7	5

[1] Group A comprised 33 individuals isolated singly or in rooms of two or three beds. [2] Group B comprised 15 individuals mixing in a common ward.

Further, *Corynebacterium diphtheriæ* was found in a higher proportion of swabs of group B (42%) than of group A (28%). It was not found in one-third of the individuals of group A, but it was found in all those of group B. Another detail not shown in the table is that *Corynebacterium diphtheriæ* was not found at the time of admission to hospital, but was found later in one of the 33 individuals of group A and in four of the 15 individuals of group B.

The evidence suggests, therefore, that the individual who is isolated, even incompletely, is less liable to cross-infection than he who mixes in the herd.

The proportion (33%) of individuals showing cross-infection in a common herd observed for seven weeks is similar to that (28%) found by Glass and Wright (1938) for individuals remaining in diphtheria wards from one to two months.

Time in Hospital.

The average time spent in hospital by 24 individuals of group A (excluding 11 from whom *Corynebacterium diphtheriæ* was never isolated) was 42·9 days; the average time spent by the 15 individuals of group B was 59·8 days. The difference suggests that the time spent in hospital by individuals of group B was prolonged by cross-infection.

One hundred and eleven other patients with very mild faucial diphtheria (carriers excluded) were observed during the same year; their average time in hospital was 44·9 days. About half were in the common ward and about half in the small rooms; but no attempt was made to isolate all of these latter, as was attempted during the experiment with groups A and B. It is disappointing that the figure for individuals under these ordinary conditions of the isolation block is but little higher than that for group A, on which isolation was imposed, although incompletely.

Tonsillectomy.

The effect of tonsillectomy was observed during the year on 41 individuals from whose throat swabs virulent *Corynebacterium diphtheriæ* was repeatedly isolated (the nasal swabs being free from the organism), and most of whom had been in hospital more than forty days. Many *Corynebacterium diphtheriæ* were isolated from nearly all tonsils examined after removal.

It was generally possible to resume bacteriological examination of the throat within one week after the operation. Of the 41 patients, 32 became "negative" within one week of the resumption of routine throat swabbing, and nine became "negative" within 10 to 35 days. Harries *et alii* (1927) have reported a similar experience. Nearly all the patients were placed, after the operation, in the one big ward together with other patients, ambulant or in bed, in various stages of the disease. Unfortunately, the types of *Corynebacterium diphtheriæ* isolated from patients after tonsillectomy were not ascertained, and therefore cross-infection has not been demonstrated; but it is suggested that some of the nine patients who did not become "negative" within one week after tonsillectomy might have done so if cross-infection could have been prevented by isolation.

General Remarks.

The practice of returning to an infected ward patients who have just been subjected to an operation which was intended to disinfect them is compelled by lack of proper accommodation. For the same reason new patients with a doubtful clinical diagnosis of diphtheria also are admitted to an infected ward. Many such patients are not suffering from diphtheria, but yet they are submitted to the risk of acquiring *Corynebacterium diphtheriæ*. The available evidence suggests that the risk is great. It is in recognition of this risk that diphtheria antitoxin is given to each patient admitted to a ward in which infective patients are being nursed, even though clinical opinion in the hospital may sometimes be opposed to the provisional diagnosis of diphtheria. The individual is thereby given temporary (passive) immunity from infection; the individual who has become infected usually possesses active immunity in addition. There is, therefore, little serious risk to patients in diphtheria wards from cross-infection by *Corynebacterium diphtheriæ*. Glass and Wright (1938) report, however, that four patients from whom *Coryenbacterium diphtheriæ* was not isolated on admission to hospital (and who, it seems, were not thought to be suffering from diphtheria) developed clinical signs of diphtheria in the hospital, and that one died. The risk is therefore serious enough; but the chief result of cross-infection is that it prolongs the patient's stay in hospital and so entails much needless expense to the hospital and exasperating loss of time to the patient.

Summary.

Individuals in hospital with mild faucial diphtheria were divided into two groups, in which evidence of cross-infection was sought for seven weeks.

No evidence of cross-infection was found in group A, in which the individuals were relatively isolated; evidence of cross-infection was found in one-third of the individuals of group B, who mixed in a common ward.

The average number of days in hospital for group A was 42·9 days; for group B, 59·8 days; for patients with similar mild disease during the same year under the ordinary conditions of the isolation block, 44·9 days.

Out of 17 nurses examined, one provided evidence that nurses may be responsible for cross-infection.

The significance of cross-infection in diphtheria wards and some of the administrative difficulties arising from unsuitable accommodation are discussed.

Acknowledgement.

My thanks are due to Dr. Eric Aspinall and to the nursing staff, whose cooperation made this experiment possible.

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SOME OBERVATIONS ON THE MASSIVE INTRAVENOUS INFUSION OF PHYSIOLOGICAL FLUIDS.[1]

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SINCE William Harvey died in 1656 many learned men have sought to unravel the mysteries of the circulation. One of these was Sydney Ringer, who studied the action of various salts on the isolated heart of the tortoise. Previously normal saline solution alone had been used as a perfusing fluid; but Ringer demonstrated that the addition of calcium and potassium greatly strengthened the heart's action.

Physicians now have a simple method of infusing large volumes of fluid into the circulation without fear of those distressing reactions of pyrexia, rigor and collapse which were frequent until a few years ago. Following the lead of American manufacturers, an Australian firm has placed in the hands of the profession flasks filled with sterile and chemically pure solutions in distilled water. These flasks can be connected to a vein of the leg or arm so that fluid drops in for minutes, hours or days, according to the need of the patient. When a massive infusion is given, two flasks are connected to a common delivery tube by a *Y* tube (Figure II). When one is empty it is clipped off and replaced by a full one, the other flask being released meanwhile. A continuous flow is thus ensured.

The body contains an abundance of water and the conservation of this is essential to life. Now that it is possible to replace fluid which has been lost in the course of dehydrating disease, many distressing symptoms, formerly dismissed as being due to "toxæmia", are no longer seen.

It is the purpose of this paper to consider a few problems of fluid deficiency, with its resulting disturbances, and to discuss some typical clinical cases.

A female patient, aged fifty years, was operated upon because of persistent vomiting caused by a diaphragmatic hernia. Six hours after operation she was cold, sweating, slightly cyanosed and semi-comatose. In any such state of circulatory collapse every endeavour should be made to ascertain what are the contributory factors. Here the history revealed that the patient had been a dietary faddist for years, taking very little protein. Further, recent vomiting had depleted her stores of fluid, food and salts; and finally the operation was accompanied both by hæmorrhage and by extensive trauma to the tissues. The post-operative treatment had consisted of the introduction *per rectum* of glucose in normal saline solution. The absorption of fluid from the rectum must practically have ceased with the onset of circulatory failure. Moreover, there is little evidence that glucose is ever absorbed from the rectum.

In view of the lack of red cells, protein, water, food and salts, it was decided to administer a blood transfusion and to follow this with a massive intravenous injection of glucose in saline solution. This was done and the result was dramatic (Figure I). The blood pressure

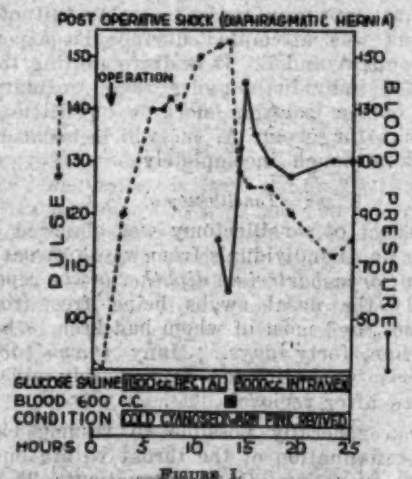

FIGURE I.

The failure of glucose in saline solution administered rectally to combat post-operative collapse. A blood transfusion followed by slow intravenous infusion of glucose in saline solution revived the patient.

rose, the extremities became warm and there was a gradual return of consciousness. At this stage biochemical investigation revealed that the blood contained normal amounts of glucose, urea, protein and sodium chloride. The patient progressed favourably for a week after this restoration, but then contracted a fulminating and fatal bronchopneumonia.

[1] Read at a meeting of the Royal Australasian College of Physicians held at Sydney in December, 1938.

Large volumes of water are constantly moving from one part of the body to another. It is of great interest to study the flow of fluid in and out of the alimentary canal. Rowntree[1] has estimated that each day approximately fifteen pints of fluid pours into the upper reaches of the alimentary canal from the secretory glands of the mouth, stomach, bowel, liver and pancreas. A similar volume of fluid is absorbed from the ileum and the colon. Mason[2] has termed this the "gastro-intestinal circulation". To this circulation there is added the daily intake of food and fluid by the mouth; and there is a corresponding loss by way of the rectum, kidneys, skin and lungs. On occasion this circulation may be broken. The victim "bleeds from his gastro-intestinal circulation". Such "bleeding" may prove fatal just as often as bleeding from the systemic circulation, although the decline may not be so rapid.

Patients suffering from intestinal obstruction due to mechanical causes, such as hernia or a peritoneal band, tend to vomit huge volumes of fæcal fluid. A similar state of obstruction may occur from paralysis of the bowel—a condition of "paralytic ileus". Here the intestine has lapsed into inertia. The abdomen is distended and silent. Large volumes of fæcal fluid are vomited in an unending stream, and the bowels do not act. Paralytic ileus is most commonly seen by the surgeon, as it tends to occur with greatest frequency after abdominal section. It may even be seen after an "interval" appendicectomy or cholecystectomy. The physician, however, is not spared, for paralytic ileus may occur in such toxic conditions as renal failure, pneumonia and septicæmia.

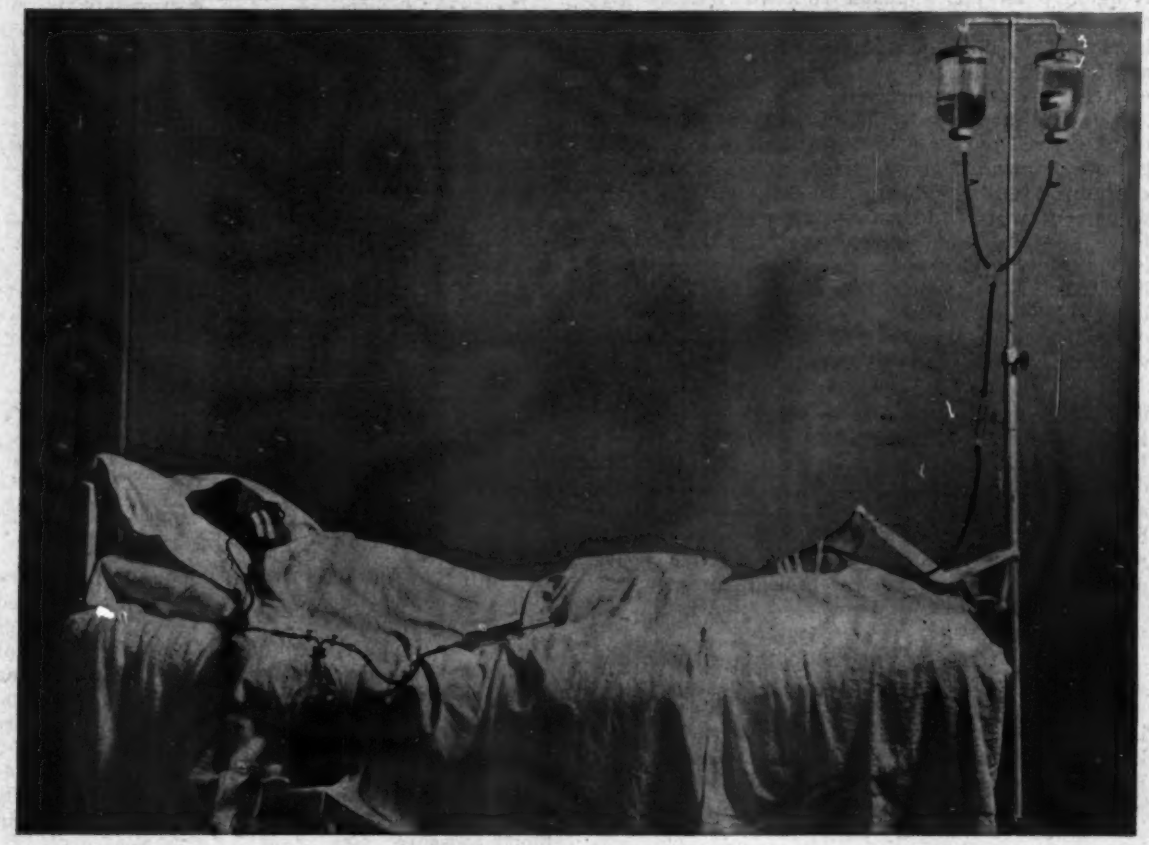

FIGURE II.

Stomach aspiration and continuous intravenous infusion in a case of paralytic ileus. The stomach tube is passed through the nose and intermittent suction is carried out by means of gentle suction with an aspirating bottle and pump. Glucose in Ringer's solution is slowly infused into the vein at the ankle to compensate for the fluid lost from the upper part of the intestinal tract.

The evolution of a successful method of treating paralytic ileus by Wangensteen[3] has probably saved more lives in the past decade than the introduction of liver therapy for anæmia. Briefly it consists of drainage of the upper portions of the alimentary canal by an indwelling tube, which is passed into the stomach by way of the nostril. The tube designed by Levine[4] is very satisfactory. The tip is similar to that of a simple urethral catheter. Lateral holes are placed at intervals of five centimetres up the tube for varying distances, according

to whether or not it is intended to allow the tube to enter the duodenum. The tube is retained in position by being strapped to the cheek, and intermittent suction is applied till the state of paralysis passes; this may be as long as a week or even a fortnight. Different methods of aspiration have been described by Wangensteen,[3] Mason[2] and other writers. A simple and effective procedure is to connect the stomach tube to a bottle such as is used for aspirating a pleural effusion (Figure II). First, the stomach is completely emptied; then aspiration is carried out at hourly intervals, and then less frequently as the stomach contents become less offensive and less copious. Should the tube become blocked, it is disconnected from the aspirator and a "Record" syringe is used to clear it by means of a little clear water syringed into the stomach. After each aspiration the patient is allowed to drink several ounces of fluid in the form of a 20% glucose solution flavoured with orange, or of broth containing sodium chloride. In this manner a large volume of water and sodium chloride is removed from the stomach and upper bowel, and the stomach is repeatedly washed out with a clear and nourishing fluid. Solid food should be avoided, as it tends to block the stomach tube. Under this treatment the distension soon becomes less and the distressing vomiting ceases.

In order to compensate for this excessive loss of fluid and chloride, and also to supply nourishment, large volumes of glucose in normal saline or Ringer's solution are poured into a vein of the leg or arm. Accurate measurement of the fluid intake and output is made and recorded on a suitable balance chart. A sufficient volume is infused to maintain a positive fluid balance. After hours, days or even a week the bowels begin to open, less fluid can be withdrawn from the stomach and the patient feels better.

Figure III represents the fluid balance in a case of paralytic ileus following an operation for chronic suppurative cholecystitis. It shows the important part played by massive intravenous infusion in maintaining a positive balance. The limited use of rectal injections is well shown. Prior to the infusion the patient was becoming drowsy, restless and collapsed. The pulse was rapid and thready, and death appeared imminent. Within three hours of the initiation of the intravenous infusion improvement set in. The vomiting practically ceased as soon as gastric aspiration was begun, but the bowels did not open until treatment had been carried out for four days. The patient made a complete recovery. It is interesting to note that Potter and Mueller,[5] in a review of consecutive cases of ileus, have found that paralytic ileus follows most commonly upon operations for appendicitis and disease of the biliary tract. It is possible that impairment of the function of the liver may play a part in the latter case.

The tone of the peripheral vascular tree is controlled by several factors. Vaso-constrictor, and to a lesser extent vaso-dilator, impulses are sent out from the vasomotor centres in the brain. Adrenaline is poured into the blood stream by the suprarenal glands, and for the most part causes widespread vaso-constriction. There are a few isolated areas, such as the lungs and the heart, where it produces vaso-dilatation. Dilatation of the capillary beds is caused by histamine and histamine-like substances, and toxins of bacterial and metabolic origin. If widespread vaso-dilatation occurs, the blood pressure may fall to an alarming degree and critical circulatory failure ensue.

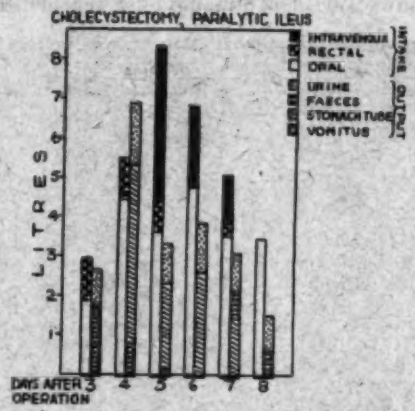

FIGURE III.

The fluid balance in a patient suffering from paralytic ileus following an operation for cholecystectomy. It will be noted that an intravenous infusion of glucose in saline solution had to be begun on the fifth day in order to obtain a satisfactory positive balance. The patient collapsed on this day, but revived soon after the infusion was begun.

In order to counteract this peripheral circulatory failure "Neo-Synephrin" (Stearns) has been injected intravenously. "Neo-Synephrin" is a substance of which the chemical formula and pharmacological action are closely allied to those of adrenaline and ephedrine. It is less dangerous than adrenaline when injected intravenously, and its action is more prolonged. Johnson[6] and others demonstrated a transient vaso-constrictor action when it was injected subcutaneously. When infused continuously into the veins it produces a more prolonged rise in blood pressure.

Sir Alan Newton has kindly allowed me to quote the history of two of his patients who benefited by the addition of "Neo-Synephrin" to the infusing fluid.

A female, aged sixty-eight years, slowly developed circulatory failure two days after resection of the caecum and ascending colon for carcinoma. A severe renal infection contributed to her toxic state. Two days after operation she was drowsy, mentally depressed and nauseated, and would not take fluids by mouth. The blood pressure was low, the extremities were becoming cold and the capillary tone was poor (Figure IV). Her urine contained albumin, pus and blood, and was decreasing in amount. A continuous intravenous infusion of 5% glucose in normal saline solution was begun at the rate of 180 cubic centimetres per hour, but although she showed slight improvement in general condition, after twelve hours the blood pressure was still low. The effect on the blood

pressure of "Neo-Synephrin" is shown in Figure IV. "Neo-Synephrin" can be sterilized by boiling and then added to the reservoir of infusing fluid. The amount of "Neo-Synephrin" used was 20 cubic centimetres of a 1% solution added to one litre of infusing fluid. It will be seen that the improvement was sustained for hours, in contrast with the transient effect of occasional hypodermic or intravenous administrations. Clinically the patient appeared to be more comfortable and mentally alert when the systolic blood pressure was above 90 millimetres of mercury. After three days of continuous intravenous infusion she was greatly improved and was able to maintain her systolic blood pressure at a level above 100 millimetres of mercury without aid.

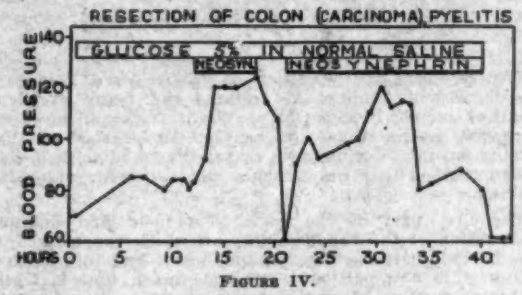

FIGURE IV.

The effect of the slow intravenous infusion of "Neo-Synephrin" (Stearns) on the blood pressure. The patient was suffering from peripheral circulatory failure following on an attack of acute pyelitis. An operation for resection of the colon had recently been performed. A slow intravenous infusion of glucose in saline solution was begun and later "Neo-Synephrin" was added to the infusing fluid. The patient was greatly improved, but suffered a relapse when the "Neo-Synephrin" was discontinued.

Figure V shows the effect of "Neo-Synephrin" in glucose and saline solution on severe circulatory collapse following a rigor.

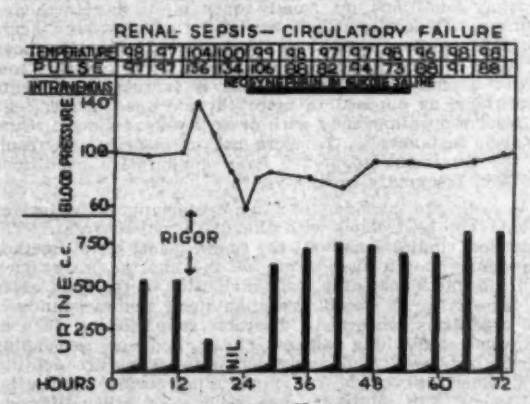

FIGURE V.

The beneficial effect on the blood pressure and urinary excretion produced by slow intravenous infusion of "Neo-Synephrin" (Stearns) in glucose and saline solution is shown. Prostatectomy had recently been performed, and this was followed by an acute exacerbation of a chronic renal infection with rigors and circulatory collapse.

A male, aged sixty-seven years, had been operated upon for an enlargement of the prostate, which had led to long-standing renal infection and finally to complete retention of urine. Two weeks after operation he had a severe rigor, which caused the blood pressure to rise rapidly. This was followed by a severe circulatory collapse. The patient was cold, semi-comatose and pulseless. The systolic blood pressure was 50 millimetres of mercury. "Neo-Synephrin" in glucose and saline solution caused a rise in blood pressure to over 80 millimetres of mercury in about fifteen minutes, and this was maintained. The solution was administered as follows. Twenty-cubic centimetres of a 1% solution of "Neo-Synephrin" were added to one litre of 5% glucose in normal saline solution and infused at a rate of 200 cubic centimetres per hour. At the same time great improvement was shown in the patient's general appearance, pulse and peripheral circulation. He eventually made a complete recovery, but only after he had been similarly treated for two similar attacks.

Lastly, I wish to refer briefly to the use of the colloid gum acacia for intravenous infusion. Following on the researches of Bayliss[7] acacia was used extensively in the Great War, and now threatens to regain its popularity. In conditions such as severe hæmorrhage, when there is a loss of protein as well as of red cells from the blood stream, the osmotic pressure of the circulating blood is diminished, and this involves a failure to restore the diminished blood volume. In order to correct the osmotic pressure gum acacia has been used. Although it is freely admitted that it is retained in the circulation for many hours and that the blood volume is maintained,[7] its use has been strongly criticized by many workers, notably Christie[8] and Studdiford.[9] Christie has demonstrated that after an infusion of gum acacia solution the oxygen content of the arterial blood is greatly reduced, the red cells being coated by the gum, a most serious event as their essential function is thus impeded.

Massive intravenous therapy is a powerful weapon. It must be used with a clear knowledge of its dangers as well as of its great advantages.

Acknowledgements.

I wish to acknowledge the generous help I have received from Dr. Charles Kellaway, director of the Walter and Eliza Hall Institute of Research in Pathology and Medicine, and from Dr. Hume Turnbull.

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Reports of Cases.

RUPTURE OF A BRANCH OF THE EPIGASTRIC ARTERY COMPLICATING PREGNANCY.

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Clinical Record.

MRS. A.B., aged thirty-nine years, was admitted to hospital during the thirty-fifth week of her eighth pregnancy. She complained of mild pain in the right side of one week's duration, associated with a severe cough. She stated that on the night before her admission to hospital the pain had become worse and had gradually increased in intensity. The pain was located in the right hypochondrium, extending from a point a few inches from the mid-line to the flank and downwards to the level of the umbilicus. Her temperature was 37·8° C. (100° F.) and her pulse rate 115 per minute. She had rather a drawn anxious expression and a sallow colour.

On examination a large mass could be felt on the right side of the abdomen. This mass appeared to be intra-abdominal; skin and fat could be moved over it, and it felt as if it were beneath the abdominal muscles. The differential diagnosis included such conditions as a fibroid growth attached to the uterus, a renal tumour and an ovarian cyst, or a tense uterus as found in accidental hæmorrhage. The clinical picture rather suggested the last diagnosis. The fœtal heart sounds were rapid and irregular, and the patient's urine contained a cloud of albumin. The systolic blood pressure, which had been found to be 140 millimetres of mercury whilst the patient was attending the out-patient department, had fallen to 110 millimetres of mercury. On pelvic examination the uterus was found to be apart from the mass. An X ray examination of the renal tract revealed a large stone in the kidney, a very dilated renal pelvis and hydronephrosis.

The ureter was then catheterized and urine loaded with pus flowed under normal tension. In view of the fact that the ureter was not actually blocked and that the position of the mass was a little too far forward for a renal tumour, it was decided that the mass was some intraabdominal lesion and was not due to an enlarged kidney. Accordingly a small pararectal incision was made. It was noted that after incision of the fascia the muscles appeared œdematous.

The muscles were separated and a large amount of old blood clot was found between the peritoneum and the rectus muscles. The amount of clot removed was sufficient to fill a large kidney dish. The cause of the hæmatoma was a rupture of a branch of the epigastric artery, probably due to the patient's violent coughing on the previous night.

Discussion.

F. H. Hobbs[1] reports a fatal case in which coughing was the initial cause of a similar condition; and J. S. Allen and Max Brodel,[2] in a very extensive review of the condition, include persistent coughing in its ætiology.

R. Mailer[3] states that spontaneous hæmatoma in women is far from common; and Hilgenreiner, in 1924, was able to collect only seven cases.

F. Norgaard states that the clinical picture is usually misinterpreted, being mistaken for a tumour of the ovary.

The case under review was rendered more interesting on account of the presence of a large renal calculus with an associated hydronephrosis, still further complicating the diagnosis. The patient made a good recovery and some months later the right kidney was removed.

References.

[1] F. B. Hobbs: "Fatal Hæmorrhage into Rectus Abdominis Muscle during Pregnancy", *The British Medical Journal*, Volume I, April 23, 1938, page 895.

[2] T. S. Cullen: "Lesions of Rectus Abdominis Muscle Simulating Acute Intra-Abdominal Condition; Hæmorrhage into or beneath Rectus Muscle Simulating Acute Abdominal Condition", *Bulletin of the Johns Hopkins Hospital*, Volume LXI, November, 1937, page 317.

[3] R. Mailer: "Spontaneous Hæmatoma of Abdominal Wall", *The British Medical Journal*, Volume I, March 28, 1936, page 637.

Reviews.

SHORT-WAVE THERAPY.

THE second English edition of "Short-Wave Therapy", by Dr. Erwin Schliephake, retains the many excellent qualities of its predecessor.[1] The translation is much improved, as the text now has but little trace of that foreign flavour so common in translations and so definitely destructive of that easy fluency necessary for enjoyable reading.

The first part of the book is a lucid and detailed explanation of the fundamentals on which the method is based. This part is crisp, comprehensive and informative, and in this new edition it contains much that is fresh, including further original researches by the author himself. The presentation and subject matter of this section are of a high grade and form an exhaustive source of information on the subject, which, as far as we know, is not obtainable as a systematized whole anywhere else in the literature. It is a complete survey of all the essentials necessary for an intelligent understanding of the subject.

The second portion, as before, relates to the clinical indications and results. This part is also expanded, so that the book as a whole is increased in size by some sixty pages. The chief interest lies in the strong claims advanced for the success of the method in the treatment of many conditions not usually amenable to more orthodox therapy. Generally it is contended that the more acute the inflammatory condition, the greater is the likelihood of relief or cure by short-wave applications. In this connexion it may be remarked that it is just these acute conditions, as opposed to more chronic ones, which tend to resolve spontaneously with or without treatment. Cure in such instances is therefore not necessarily the result of the treatment adopted. Even suppurative conditions are said frequently to resolve.

According to the older teaching, acute suppurative lesions should not be treated with the diathermy current. The extended indications of the new short-wave method apparently lie in the nature of the shorter wave itself and the consequent differing distribution of the heat, aided perhaps by some specific reaction upon the resistance of the organisms concerned. Thoracic suppurations, such as empyema and lung abscess, are cited as responding particularly well, and many successful cases are detailed and illustrated by X ray films taken before and after treatment. In addition, case reports of other different conditions are brought forward, and, though some of the successes claimed are not completely convincing, the impression remains that some remarkable results have been obtained.

The book as a whole is a well-reasoned exposition of the subject, free from extravagance and filled with interesting and suggestive detail. The production is of the same high quality as the contents.

[1] "Short-Wave Therapy: The Medical Uses of Electrical High Frequencies", by E. Schliephake; authorised English translation by R. K. Brown, B.A., M.D., D.P.H., from the third German edition, with a foreword by H. P. Cumberbatch, M.A., M.B., B.Ch., F.R.C.P., D.M.R.E.; Second English Edition; 1938. London: The Actinic Press Limited. Medium 8vo, pp. 396, with illustrations. Price: 21s. net

The Medical Journal of Australia

SATURDAY, JUNE 10, 1939.

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APPENDICITIS.

DISCUSSIONS on appendicitis in these columns have dealt chiefly with methods of treatment. Such questions as the diagnosis of the condition, its relation to other disorders of the intestinal tract, the importance of early operation and the indications for the institution of drainage have been considered. Of the causation of appendicitis we know comparatively little. We know that its immediate cause is a bacterial infection, but beyond this information is indefinite. Certain habits of life predisposing to intestinal stasis, as well as the constituents of modern diets, have been incriminated, apparently with some justification; but the matter is still undetermined. Until some avoidable cause is shown to exist, there is little hope of material reduction of the ravages of this latter-day disease. For this reason particular interest attaches to a recent statistical study carried out under the ægis of the Medical Research Council of Great Britain by Dr. Matthew Young and Mr. W. T. Russell.[1]

The first conclusion to be drawn from this study is that the incidence of appendicitis has increased. Young and Russell are cautious in making this statement. Comparison of records from Great Britain, Germany and America for recent and earlier periods reveals a great decline in "case fatality" from the disease. This decline in case fatality has been accompanied by an apparent increase in its incidence. Caution, however, as Young and Russell point out, must be exercised in the drawing from hospital statistics of inferences regarding the magnitude of this increase, since the change may be partly due to the fact that admission to hospital has become more common. For example, it would be dangerous to draw a general conclusion from the discovery that the number of patients treated in 75 American hospitals during 1923 was 18,194, and that in 1932 the number was 26,261, an increase of 44%. Young and Russell hold that the relative constancy of the mortality rate in Great Britain and its increase in Germany, with the accompanying great reduction of the case fatality rate, can be satisfactorily explained only by an increased incidence of appendicitis. This conclusion is supported by the fact that amongst the insured population of Scotland, the only instance in which published statistics of the incidence of the disease on a national scale exist, the annual case death rates have increased and the increase is not correlated with an upward trend in the general sickness rate. Turning to the mortality rate, we find that in England and Wales as a whole it has shown no definite trend upward or downward during the last twenty years. There has, however, been a curious change in the age distribution of the deaths. The mortality rates at ages nil to five and over forty-five years have increased and at other ages there has been a considerable decline. The same, we are informed, is revealed by the American statistics. The mortality, moreover, among males is higher than among females, the excess in the former being of the order of 25% to 30%. Among women the mortality is heavier among single than among married women over fifteen years of age. In England and Wales the mortality rate for rural districts differs but little from that for urban districts; in Germany the town rate seems to be greatly in excess of the country rate. In its relation to the social class of the

[1] "Medical Research Council of the Privy Council. Special Report Series, Number 233: Appendicitis, a Statistical Study", by M. Young and W. T. Russell; 1939: His Majesty's Stationery Office. Royal 8vo, pp. 79. Price: 1s. net.

patient the mortality rate shows striking variations. It is greatest in the so-called upper classes and declines progressively with the social grade—the mortality among the wealthier members of the community is at least two and a half times greater than among the very poor. It is pointed out that over-indulgence in food or in particular types of food is probably more common among the well-to-do, and that possibly the practice of the taking of laxatives or purgatives for any type of abdominal pain is commoner among them. This is regarded as little more than suggestive, and attention is drawn in the Medical Research Council's preface to the similarity of the histories of mortality from appendicitis and from *diabetes mellitus* among women during the war and post-war periods. The mortality from both diseases among women at the higher ages fell when food restrictions were imposed during the war years, 1915 to 1918, and increased subsequently. Many other aspects of the subject are dissected statistically, but reference will be made in this place only to the psychological factor. The suggestion that many conditions diagnosed as appendicitis may not be true appendicitis but may merely simulate it and depend on the psychological make-up of the individual, is not seriously regarded. That the psychological factor has in recent years influenced the recorded incidence "is of relatively small, if not negligible, importance". Of the figures supporting this view, it may be of interest to refer to those from Edinburgh hospitals, reported by Wevill and Wallace—in only 487 or approximately 6% of 8,000 cases classified as acute appendicitis was no disease of the appendix found at operation to account for the patient's symptoms.

Extensive though this research has been, it does not lead us very far on our road to the discovery of the causation of appendicitis. Young and Russell are rightly most cautious in drawing dogmatic conclusions. They refer to the contention of Rendle Short that the increased incidence of appendicitis in Great Britain has been mainly due to the change in modern diet—the reduction in cellulose and fibre content and the increased consumption of imported foods, especially meat. In support of these contentions it has been stated that appendicitis rarely occurs in institutions because of the plain fare of the inmates, that it is rare among primitive people living on a diet with an abundance of cellulose, and that it becomes common if such people change to European food. Young and Russell describe these observations as "based on general impressions and not on adequately controlled observations". All the same, though they do not make the statement in so many words, they would apparently, if they could, make people live on a Rendle Short diet. They refer to propaganda for the adoption of this type of diet and add that "under existing conditions of modern life it seems improbable that sufficient change in dietary habits will be introduced to influence in any appreciable degree the incidence of appendicitis". They also mention the favourable results achieved at Philadelphia over a five-year period by a propagandist campaign to promote the early admission of appendicitis patients to hospital and the avoidance of laxatives in the presence of symptoms suggestive of inflammation of the appendix. An account of this commendable effort (which might well be adopted in Australia) will be found in THE MEDICAL JOURNAL OF AUSTRALIA of July 7, 1934, at page 19. The general conclusion—and no other could be drawn from this investigation—is that the only hope of effecting a further reduction in the mortality rate of this disease is to encourage, by the education of the public, earlier entry to hospital and the avoidance of purgatives in the presence of any suspicious symptoms. If we accept this conclusion we must presume that medical practitioners are thoroughly conversant with the signs and symptoms characteristic of the early stages of appendiceal inflammation. In any case Young and Russell's work has again directed attention to our ignorance of an important and widely spread condition, which may be largely the result of errors in environment and modes of living. The Medical Research Council is of this opinion, and it insists at the conclusion of its preface that investigation should be directed to the discovery of the "special causative harmful influences and mistakes which civilisation is unknowingly embracing in its advance".

Current Comment.

EPIDEMIC DROPSY.

EPIDEMIC DROPSY was for many years regarded as a deficiency disease. It was even at times confused with beri-beri. Within the past few years its distinction from beri-beri has been clearly established, and a considerable amount of evidence has accumulated to the effect that it is not caused by vitamin deficiency. At one time or another it has been suggested that the ingestion of mustard oil or some adulterant of mustard oil might be the main ætiological factor. Mustard oil is used freely in many parts of India in the cooking of rice. It has been pointed out that persons who use mustard oil are far more liable to the disease than other persons in the same community. In 1926 Sarkar suggested that argemone oil, which sometimes occurred as an adulterant of mustard oil, might be responsible. In 1935 Chopra and Chaudhuri propounded a theory that the disease was caused by infection with organisms of the *vulgatus* group. In households where epidemic dropsy occurred they found the rice to be heavily infected with these organisms, and they found that the serum of patients caused agglutination of certain strains of the organism at a high titre. The interest in mustard oil was revived by work carried out by Lal and Roy, reported in 1937. A number of volunteers were given food cooked in samples of mustard oil that had been used by persons suffering from epidemic dropsy. These volunteers became affected, whereas no member of a control group, fed with pure mustard oil, had any symptoms of the disease. Lal and Roy did not recognize the impurity in the oil that seemed to be responsible.

Further investigations have been made by R. N. Chopra, C. L. Pasricha, R. K. Goyal, S. Lal and A. K. Sen.[1] They examined samples of mustard oil obtained from houses in which outbreaks of epidemic dropsy had occurred; in the majority they found traces of argemone oil. A plant known as *odissimari*, which bears seeds very like mustard seeds, grows freely in many mustard fields in India. It has been shown to be identical with *Argemone mexicana*. Argemone oil must therefore occur frequently, either by accident or design, as a contaminant of mustard oil.

Chopra and his co-workers gave food cooked in mustard oil and argemone oil (both expressed in the laboratory) to five volunteers. To four others they gave food cooked in mustard oil (contaminated with argemone oil) obtained from households where outbreaks of epidemic dropsy had occurred. The members of a third group were given food cooked in pure mustard oil. None of the members of this group suffered from symptoms of illness. All members of the first group became affected with œdema and other symptoms indistinguishable from those of epidemic dropsy. Two members of the second group suffered from what appeared to be epidemic dropsy; the other two were not affected. One of the latter two was given the contaminated oil for a shorter period than his fellows, because of the occurrence of a fever; the other took less rice than the rest of the volunteers, though he took a similar amount of oil. The evidence suggested that argemone oil had a cumulative effect; mustard "oil used at the time of an outbreak can be pure and innocuous but it is the oil used some days before the onset of symptoms which is responsible". Argemone oil becomes harmless to laboratory animals when it is heated to 240° C. It is not known whether the harmful substances are destroyed by this heat, or whether they are present in the distillate.

As these workers remark, it cannot be asserted that the condition produced experimentally by the ingestion of argemone oil is identical with epidemic dropsy; but the evidence is so strongly suggestive as to be almost conclusive. The next step might well be the elimination of *Argemone mexicana* from certain mustard fields, and observation of the health of people who use the mustard obtained from these fields, as compared with the health of neighbouring communities. One by one the mysterious eastern diseases are being robbed of their mystery by systematic research, not by one man or a few men, but by many men, of perhaps many generations. The solution (admittedly not yet complete) of the riddle of epidemic dropsy is a striking example.

[1] *The Indian Medical Gazette*, April, 1939.

ACQUIRED SYPHILIS IN CHILDREN.

FRANK R. SMITH, JUNIOR, remarks that an important matter is the extent to which the ordinary household contacts with a person who is suffering from early syphilis enhance the danger of infection.[1] He states that data on this point which can be subjected to quantitative analysis are difficult to obtain. If living in a home with a potentially infectious syphilitic patient constitutes an added risk of infection, then infection of children in households in which adults are suffering from primary or secondary syphilis should not be uncommon. However, it is well known that it is uncommon to observe acquired syphilis in children. The fact of its occurrence at all suggests that ordinary household contact may lead to transmission of the disorder. Smith considers that in many recorded instances, particularly prior to the common use of the Wassermann test, probably congenital syphilis with unusual lesions or delayed clinical signs was confused with acquired lues. Smith has collected a series of 125 children with acquired syphilis, representing a search for cases extending over a year and necessitating a journey from Boston to New Orleans. When a personal visit was impracticable a form was forwarded con-

[1] *American Journal of Syphilis, Gonorrhœa and Venereal Diseases*, March, 1939.

taining the requisite queries. An upper age limit of ten years was chosen, as, after that age, the sexual precocity and promiscuity of many children, particularly negroes, make the usual method of infection by sexual contact so common that the disease is similar in every respect to acquired syphilis in young adults. Accordingly, 90 instances of acquired syphilis in children between the ages of eleven and fifteen years, observed in the Johns Hopkins Hospital, were excluded. These facts are rather staggering to contemplate.

The 125 cases tabulated were not all of those occurring in the localities indicated. There is a tendency to consider all instances of syphilis in children as congenital in the absence of outstanding features of the acquired disorder. Many cases were discovered only by painstaking investigation to have been acquired when they had been recorded as congenital. All cases were excluded in which there was doubt. When by signs, symptoms and clinical history the diagnosis was not obvious the following criteria were met: (i) The child was known to be clinically and serologically "negative" for syphilis previously. (ii) Siblings did not have congenital syphilis. (iii) If the infection was acquired from a parent after birth, that the mother was non-syphilitic at the time of the birth of the child and had received no antisyphilitic treatment to alter her blood serological reaction. Smith considers it certain that some children with acquired early syphilis are never seen by a physician, as the families regard the condition as a mild exanthem not needing medical care. As the material in this series was from both northern and southern States, negro and white children were almost equally represented. Of the 45 sufferers from the Johns Hopkins Hospital, 12 were white and 33 coloured. In this hospital new admissions to the general pædiatric clinic are 65% white and 35% coloured. This contrasts with the race distribution of the cases of acquired syphilis, which shows 27% white and 73% coloured. Acquired syphilis in children is more common in girls than boys, in the proportion of almost 2 to 1. Smith states that this preponderance amongst females is mainly due to the fact that they are the objects of attempts at sexual intercourse by older children or adults.

Extragenital chancres are more common in small infants. As the children grow older and more sexually active the genitally placed chancres increase. When the disease is acquired by sexual contact females are infected two and a half times as frequently as males. Non-sexual transmission is equal in the sexes. It was not always possible to trace the immediate contact in every patient. The source might not be known or, if known, withheld. Rape was assigned as a cause in four patients and pæderasty in two. In one instance the father acquired syphilis and transmitted it to the housekeeper who was caring for the family while the wife was in hospital. From the housekeeper one child acquired an extragenital chancre. When the wife returned home she became infected, but the new-born infant had neither congenital nor acquired lues. When a daughter was infected by a diseased father the method of transmission was usually through some sexual practice, but infection of children by diseased mothers was by non-sexual contact. Some of the cases recorded by Smith are revolting. No instance of the so-called binary syphilis was observed in this series. E. Hoffman and others have mentioned the possibility of the occurrence of mixed forms of congenital and acquired syphilis in children. He has quoted several instances and considers that they represent superinfection. The location of the primary lesion was ascertained in 77 of the 125 patients. Extragenital chancres were frequent and 90% were found about the face and neck, which are likely points of contact with mouth lesions of the infecting person. Chancres of the anus are the result of attempts at intercourse or abnormal sexual practices. Smith remarks that the almost equal number of males and females infected extragenitally contrasts with the much greater number of females infected genitally. The chancre, particularly when genitally situated, is often smaller than in adults, but has the customary form of a hard indurated erosion with elevated borders. On two occasions a primary lesion was found on the cervix while the patient was being treated for gonococcal vaginitis. Condylomata about the anus or perineum were common in females. The skin lesions were generally maculo-papular or annular. There were no bullous eruptions. The generalized lymphadenopathy was slight. There was no pronounced enlargement of the liver and spleen, as in congenital syphilis. Four patients had alopecia. Buccal mucous patches were frequent. Periostitis, pseudo-paralysis and snuffles were not noted, this contrasting with early congenital syphilis. Gummata were not recorded nor were lesions of the cardio-vascular system. The routine treatment consisted of courses of neo-arsphenamine, alternating with intramuscular injections of bismuth subsalicylate. The immediate results of treatment were good. Lesions healed promptly and early serological reversal was generally obtained. Smith considers that the rapidity of serological response is a valuable diagnostic aid in the differentiation of acquired from congenital syphilis. Children tolerate antisyphilitic drugs better than adults, and no severe reactions were seen in this group. Smith records, in this material, nine cases of syphilis in children in which the disease resulted from transfusion. He refers to the writings of J. MacQueen regarding *syphilis insontium* in Palestine, and of E. H. Hudson concerning *Bejel* or non-venereal syphilis. He concludes his article by stating that casual contact even with a person in the active stage of syphilis rarely results in infection of the child. The risk of the acquirement of syphilis by the ordinary contacts between individuals living in the same house, such as is encountered with food-handlers, nursemaids and servants, seems to be small. When, however, the contact is intimate, as in the case of young children

with their mother, the risk is sufficient to have a practical significance. Infection by playmate contact requires various forms of secretive infantile sex practices rather than the ordinary conditions of play.

THE SEARCH FOR SUBSTITUTES FOR MORPHINE.

THE march of pharmacological science might well be epitomized in the three words poppy, opium and morphine, and to these must now be added the synthetic alkaloid. The poppy, more strictly speaking the variety *Papaver somniferum*, has long since won its place in literature by reason of being the source of the drug which more effectively than any other brings sleep and relief from pain. Opium, its comparatively crude but useful product, next yielded its more or less standardized preparations, and itself enriched our vocabulary with another word in general use. Finally morphine and its allied alkaloids represent the last step in the accurate work of the pharmacologist in producing the active principles. But even this, probably the most beneficent of anodynes known to man, has its drawbacks and dangers. Even apart from overdosage, idiosyncrasy is not unknown, and care must be taken to avoid the "drowsing away to poppied death" of which the poet speaks. Far more dangerous and insidious is the risk of addiction. That this has a physical basis is undoubted, for all the derived alkaloids have not an identical effect, regarding either their immediate action or their tendency to establish habit. De Quincey was probably the most famous exponent of the euphoric qualities of opium. During "a debauch of opium", as he candidly called it, every pleasure was enhanced; anxieties were smoothed by "a halcyon calm, tranquillity that seemed no product of inertia, but as if resulting from mighty and equal antagonisms; infinite activities, infinite repose", sensuous enjoyment, as of music, was a "golden epiphany". But every practitioner of medicine should read the later and sadder pages of this famous record, with its ineffectual rationalization, its conscious and unconscious revelation of the terrible penalties of addiction. Many others before and after De Quincey have paid the price of this physical and mental dependence upon a narcotic drug, and many of those in whose hands rests its legitimate employment have longed for some drug equally potent in relieving pain and restlessness, but free from these disadvantages.

That there is a definite relationship between the quality of causing physical dependence and the actual chemical structure of these drugs seems likely, for all the clinical evidence points to some subtle but destructive change in the body tissues by prolonged addiction to morphine or heroin. This belief is held by Nathan B. Eddy, who has recently given an account of an investigation which has been carried out in America during the past ten years, sponsored by the National Research Council of the University of Virginia, the University of Michigan and the United States Public Health Service.[1] No fewer than 47 persons have been concerned in the carrying on of this work, whose object is the production of a non-addicting analgesic. This research utilizes the latest modern method of the chemist, that of synthesis, whose full results we cannot yet imagine. Two plans have been followed. In the first the alkaloids whose structural formulæ were related to morphine were examined, their nuclear skeletons were modified by various side groups, and efforts were made to discover the relations of these components of the product to the known effects of morphine. In the second method synthetic compounds were made containing unrelated or distantly related nuclei; obviously this would entail great labour and be subject to even greater uncertainty as to the results. Eddy points out that the structure of morphine and also of the naturally occurring alkaloid codein (which is closely related to morphine) allows the existence of eight isomeric compounds of each. All these have been examined with regard to their effective dose and toxicity, particularly regarding their effect as respiratory depressants, and in addition various substitutions in the morphine nucleus have been made which have given rise to some promising compounds. Such investigations are not simple or rapidly made, and they are made more difficult by the fact that in some animals morphine has a convulsant effect not observed in man. Methyldihydromorphinone is one of these compounds which is now being submitted to clinical trial. In man it is said to be twice as effective an analgesic as morphine, with a duration of action approximately the same, while no emetic action or sign of respiratory depression has been observed. This drug has been used in human morphine addicts, and the very brief duration of what Eddy calls dependence satisfaction leads these workers to hope that it would not readily cause addiction. There are many other promising possibilities; the investigators have pointed out that only trial can show whether it is one or more of the peripheral groups of the morphine molecule or the phenanthrene nucleus which is really responsible for the characteristic properties of the alkaloid, and both synthetic routes must be explored in this search for the ideal narcotic. It is interesting to read that a score of substances have so far been submitted to clinical trial; of these, methyldihydromorphinone seems the most promising.

Probably no era in medical science has been so exciting as the present. Nobody knows when the most drab of pharmacological researches may bourgeon into a brilliant gift to mankind; but the industrious workers concerned must know that if they can ever discover the perfect anodyne, potent, safe and relatively free from the perils of addiction, it will not be greeted with the silent wonder of "stout Cortez, when with eagle eyes he stared at the Pacific", but with the immediate acclaim of the medical world.

[1] *The American Journal of the Medical Sciences*, April, 1939.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Hæmatometra.

PHINEAS BERNSTEIN AND ROBERT WALTER (*American Journal of Obstetrics and Gynecology*, January, 1939) observe that the subject of hæmatometra is briefly described in most text-books and only occasionally referred to in the literature, and so merits presentation. The incidence was 0·1% among all gynæcological admissions to Mount Sinai Hospital during a twenty-year period. The authors consider this group of nineteen patients. In eleven cases the hæmatometra was due to a congenital condition—imperforate hymen in ten and a bicornuate uterus in the other one. The patients with imperforate hymen were all between the ages of twelve and sixteen years, had never menstruated, and complained of pain either low in the back, abdominal or suprapubic. Four had urinary symptoms, two had pain, tenderness and rigidity low on the right side of the abdomen. The authors remark that in spite of the fact that the patients are afebrile and not acutely ill, they are not infrequently operated upon for appendicitis. The signs are a bulging, blue, imperforate hymen and a distended vagina, upon which may be superimposed a cystic uterus with lateral tender sausage-shaped masses. Bernstein and Walter hold that the malformation occurs during development in the lower third of the vagina, where the lower ends of the Müllerian ducts meet the vulval ectodermal process. The order of dilatation above the obstruction is, first, vaginal, then vulvo-perineal followed by corporal, then cervical and finally tubal. Hæmatoperitoneum may result; it occurred twice in this series and was associated with endometriosis. The end stage of hæmatometra is compression of glandular, vascular and structural elements. The usual amount of red or brownish treacle-like fluid is 500 to 1,500 cubic centimetres. In nine cases there was acquired stenosis, due to one of the following causes: injury sustained during delivery followed by infection, cervical fibroadenoma, cauterization of the cervix, the use of X rays, involutional cervical changes. Of these causes infection following obstetrical injuries was the most common. There is usually a history indicating a cause for these acquired lesions. The stenosis is not at first complete and the condition is not usually recognized until superadded infection is established. A correct preoperative diagnosis is rarely made and the lesion is confused with appendicitis, double uterus, normal pregnancy, abortion and ectopic gestation. The treatment of gynatresia is always surgical. The extent and method of the surgical procedure depend on the site and type of atresia, the patient's age, the necessity for preserving the child-bearing function, and the presence or absence of infection. The prevention of secondary infection and the establishment of free but slow drainage are the fundamental principles involved. Simple procedures were usually adequate for the younger patients of this group. For imperforate hymen complete excision is practised; pelvic examinations after operation are not performed. The patients are nursed in Fowler's position for fourteen days.

Studies on Pregnandiol.

ROBERT B. WILSON, LAWRENCE M. RANDALL AND ARNOLD E. OSTERBERG (*American Journal of Obstetrics and Gynecology*, January, 1939) have studied the excretion of pregnandiol in various conditions. Pregnandiol is a saturated alcohol and differs but little from the unsaturated diketone progesterone. Pregnandiol is the only known excretion product of the *corpus luteum* hormone, progesterone, in the urine. It is considered that the amount of pregnandiol in the urine is an index to the function of the *corpus luteum*. The author's estimations of the amounts of pregnandiol in the urine of presumably normal women during the luteal phase of the menstrual cycle correspond to those made previously by Venning. On the twelfth day before menstruation approximately one milligramme of pregnandiol is excreted in twenty-four hours. This figure rises to slightly over three on the eighth day before menstruation and begins to fall on the fourth day before menstruation, to reach a minimum of less than one at the onset of the menstrual flow. The authors then studied the relation of pregnandiol excretion to the phases of the menstrual cycle as revealed by endometrial biopsy. Endometrial biopsy also allowed them to differentiate those patients whose endometrium, as well as being in the late proliferative phase, also showed cystic changes in the glands. The authors conclude that the endometrial response to progesterone is a variable factor and that it may ultimately be shown that the normal hormonal function is more dependent on the relation between the amounts of œstrogenic hormone and progesterone than upon the actual amounts of either. They infer that the œstrogenic hormone secreted by the *corpus luteum* is essential to the development and function of the endometrium during the luteal phase of the menstrual cycle. They consider that an evaluation of the function of the *corpus luteum* is much more accurate when several determinations of pregnandiol can be made, and that normal values for pregnandiol and a normal menstrual history may be entirely misleading unless endometrial biopsy is performed. In pregnancy they found that pregnandiol was excreted either in normal amounts or in amounts in excess of those found during the luteal phase of the menstrual cycle, so that pregnandiol excretion in significant amounts was probably a sign of pregnancy. The amount of pregnandiol excreted on the fourth day *post partum* was more than one milligramme in 55% of cases, above two milligrammes in 34%, and above four milligrammes in 18%. The authors conclude that there is some luteal function after delivery.

The Treatment of Acute Pulmonary Œdema.

H. DÖRR (*Monatsschrift für Geburtshülfe und Gynäkologie*, September, 1938) discusses the value of venesection in acute pulmonary œdema. His patient suffered from ruptured ectopic pregnancy, and autotransfusion was performed. Acute pulmonary œdema developed within twenty-four hours and was probably due to an allergic reaction. Recovery followed three small venesections. This complication shows the possible risks of autotransfusion, which should be employed only in exceptional circumstances.

The Circulation during Pregnancy.

C. S. BURWELL *et alii* (*Archives of Internal Medicine*, December, 1938) have studied the alterations from normal which take place in the maternal circulation during pregnancy. They find the chief of these to be: (i) an increased cardiac output per minute by as much as 50%; (ii) a decreased arterio-venous oxygen difference; (iii) a rise in the venous pressure in the lower extremities; (iv) a loud bruit over the placental site; and (v) an increase in the blood volume. They conclude that these changes are to be ascribed to two main mechanisms, a great arterio-venous "shunt" in the placenta and obstruction to venous return by the enlarged uterus. A summary in English is given of a treatise by R. Spanner on the vascular system of the placenta.

OBSTETRICS.

Studies in Pelvic Arrests.

W. E. CALDWELL, H. C. MOLOY AND D'ESOPO (*American Journal of Obstetrics and Gynecology*, December, 1938) carry their investigations of the female pelvis a stage further. In the present article an investigation is made of mechanisms used in forceps delivery in relation to pelvic type. From an analysis of the method of delivery in the five hundred cases under review, it was found that if the films were viewed in the precision stereoscope a more accurate idea of the pelvis could be obtained than by any method of mathematical calculation of diameters. For example, in

anthropoid types the long antero-posterior diameter may compensate for the narrow interspinous diameter; but in android types there is less compensating space in the sagittal plane or in other regions of the pelvis, and accordingly the visual study of the stereoröntgenograms represents the best method in the examination of the pelvic architecture. The mechanism depends on the type of pelvis. The transverse oval shape is predisposed to a transverse mechanism throughout the pelvis; less trauma to mother and child results if the head is made to descend to lower levels in the transverse position. Separation of the symphysis, stillbirth, an infant suffering from shock or serious injury to the soft parts has occurred by failure to maintain this transverse mechanism by premature attempts at anterior rotation in certain android and flat pelvic types. Four cases are selected from the stillbirths that occurred in this series, to illustrate the following principles of good mechanisms in delivery. Forceful attempts at anterior rotation in flat and certain android pelvic types should not be made; the transverse mechanism to lower limits should be encouraged in these forms. Forceful attempts at anterior rotation in low occipito-posterior arrest in the extreme anthropoid pelvis are equally dangerous; the head should be elevated and rotated at a higher level or brought to a lower level in the occipito-posterior position.

Pyelectasis of Pregnancy.

S. R. WOODRUFF AND A. H. MILBERT (*The Journal of the American Medical Association*, October 29, 1938) report urographic studies of sixty normal women during and after pregnancy. Nearly all these women developed slight or gross pyelectasis, which was no longer present shortly after their delivery; none had any urinary infection. The authors believe that pyelectasis of pregnancy is physiological, and that pyelitis of pregnancy usually arises from a morbid condition of the urinary tract antedating pregnancy.

Diagnosis of the Occurrence of Toxæmia of Pregnancy by Examination of the Unknown Placenta.

R. A. BARTHOLOMEW AND E. D. COLVIN (*American Journal of Obstetrics and Gynecology*, December, 1938) record the relationship between the lesion found and the clinical diagnosis in a series of 100 consecutive unknown placentæ. As a result of a gross examination they were able to diagnose the occurrence of severe toxæmia in 90% of the 100 unknown placentæ examined. They were of the opinion that placental infarcts are definitely associated with the more acute types of toxæmias. Placental tissue contains twice as much arginine as any other tissue, and this is the probable explanation of the specific eclamptogenic character of placental autolysate. This action is brought about by the formation of guanidine from arginine, and the guanidine is known to raise blood pressure and produce convulsions. The authors came to the conclusion that the following chain of circumstances is responsible for eclampsia: first there is trauma to placental arteries by fœtal movements; then localized cholesterol changes take place, more especially when hypothyreoidism is present and when the patient is receiving a diet rich in cholesterol; then, as the result of trauma, placental infarcts are formed, which lead to placental autolysis, and the autolysate, being rich in arginine, the precursor of guanidine, produces the typical eclamptic condition.

Post Partum Necrosis of the Anterior Pituitary.

H. L. SHEEHAN in 1937 showed that massive necrosis of the anterior pituitary is a relatively frequent *post mortem* finding in patients who die during the puerperium after a delivery complicated by collapse or severe hæmorrhage. H. L. Sheehan and R. Murdoch (*The Lancet*, July 16, 1938) give the histories of eight patients who had severe *post partum* hæmorrhage in a confinement. Their symptoms varied in relation to the amount of necrosis of the anterior pituitary that had taken place. Some manifested lassitude and weakness and disturbance of menstruation from scanty menstruation to complete absence. Sections of a gland taken from one patient who died in a subsequent pregnancy are illustrated. Patients who have had a *post partum* necrosis of the anterior pituitary may subsequently become pregnant if genital atrophy has not taken place. Such a pregnancy usually brings great improvement or even complete and permanent cure of symptoms of pituitary insufficiency. If, however, the delivery is accompanied by hæmorrhage or collapse the improvement is partly or entirely lost.

Uric Acid Metabolism in Eclampsia.

J. F. CADDEN AND H. J. STANDER (*American Journal of Obstetrics and Gynecology*, January, 1939) studied the amounts of uric acid in the blood and urine of five patients with eclampsia. Estimations were made in milligrammes per hundred cubic centimetres daily during the stage of convulsions and every few days during the puerperium and were plotted on a graph. The study showed that the amount of uric acid in the blood was increased, that uric acid excretion was unimpaired, and that the concentration factor was not below normal limits during the height of the disease. They conclude that the increased amount of uric acid in the blood in eclampsia is due to an impairment of uric acid destruction in the liver and not to a decreased excretion by the kidneys. The increase in the amount of uric acid in the blood occurs early in the disease and is the earliest perversion of metabolism noted in the development of eclampsia. The authors also consider that this study has brought forward additional evidence that eclampsia is the outcome of a metabolic disturbance.

Uterine Inertia.

T. N. A. JEFFCOATE (*The Journal of Obstetrics and Gynæcology of the British Empire*, December, 1938) discusses uterine inertia, with special reference to the results of treatment with œstrogenic substances. He regards parturition not as a single isolated event, but as the climax of a process which begins with ovulation and ends with involution. The physical development of the uterus during pregnancy is dependent on the effects of *corpus luteum* and œstrogenic hormones. Progesterone is inhibiting, while the œstrogens are activating. In the human female during pregnancy the œstrogenic hormone arises in the placenta, and so the fœtus controls the determination of its own intrauterine existence. Œstrogenic principles acting on uterine muscle not only increase its vascularity, metabolism, growth, tone and sensitivity to oxytocic agents, but also control the coordination of uterine contractions. As pregnancy advances the amount of œstrogenic hormone in circulation increases, but it remains mostly in an inactive form until the later weeks, becoming activated just prior to labour. Inertia which is not the result of mechanical obstruction to delivery may be caused by a relative insufficiency of œstrogenic hormone To test the effect of œstrogenic hormone administration, only patients with inertia of long standing and characterized by complete or almost complete cessation of the contractions were accepted. Thus only 88 patients out of 1,200 were selected, of whom only 16 had been less than twenty-four hours in labour when treatment was commenced. Ordinary methods of treatment, such as enemata, catheterization and sedatives, were always tried first. Treatment was successful in 50 out of 88 cases, labour being completed spontaneously in 27. In 34 the treatment failed. When mechanical factors obstructing delivery, such as disproportion or malrotation of the head, were absent, success was obtained in 74%. The treatment is free from risk to both mother and child. The author considers that the prophylactic injection of œstrogenic hormone is also indicated when labour may be reasonably expected to be prolonged and difficult. Nevertheless the importance is stressed of regarding every patient exhibiting feeble or irregular uterine contractions in labour as suffering from an error in the mechanical relation between the presenting part and the birth canal.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on April 19, 1939, at the Women's Hospital, Carlton. The meeting took the form of a number of clinical demonstrations by members of the honorary staff. Part of this report appeared in the issue of June 3, 1939.

Gynæcological Apparatus.

DR. A. SHERWIN, assisted by Miss Horwood, demonstrated the various types of apparatus used by the hospital for the treatment of pelvic infections. These consisted of Leiter's coils, an electric tent, and machines for ultra-violet rays and short-wave therapy and for diathermy. Patients shown and case histories reported illustrated acute and chronic salpingitis and pelvic cellulitis. Dr. Sherwin said that records of this expectant treatment showed that operations for acute salpingitis had become comparatively rare. An increasing percentage of patients were reporting who had become pregnant after leaving hospital.

Ovarian Cyst.

DR. EDWARD WHITE's first patient was a married woman, aged forty-four years, who had one child, aged twelve years. She had noticed a rapid swelling of the abdomen during the previous four months and had lost weight. Examination revealed a large ovarian cyst entirely filling the abdominal cavity. This was thought to be an adeno-carcinoma. Operation and deep X ray therapy were advised.

Perineal Tear.

Dr. White also showed a patient, aged twenty-eight years, who had a complete perineal tear, which was very infected. Three weeks earlier she had had her first confinement in the country. The fœtus had presented by the breech, with legs extended. Dr. White said that it would be advisable to allow the wound to clean up and to perform a complete repair operation after three months.

Dr. White showed another patient who had had a complete perineal tear after her confinement, many years earlier. Fifteen years later Dr. White had performed a complete perineorrhaphy with a satisfactory result.

Puerperal and Abortal Sepsis.

Dr. White demonstrated the temperature charts of several patients with acute puerperal and abortal sepsis, from whom hæmolytic streptococci had been cultured; he also showed the temperature charts of patients suffering from acute pyelitis of pregnancy and from *Bacillus coli* infections. He said that in every instance chemotherapy with sulphanilamide was followed by good results.

Cardiac Disease Associated with Pregnancy.

PROFESSOR R. MARSHALL ALLAN presented two patients with cardiac disease associated with pregnancy. The first patient, aged twenty-two years, who had had two children, was suffering from auricular fibrillation. She had had rheumatic fever at the age of fourteen years. She had had a normal confinement three years previously; this had been followed by dyspnœa five months later. Since then she had been in and out of hospital on many occasions. During this period she had been receiving treatment with digitalis and had had several attacks of congestive heart failure. About two years earlier she had apparently had a cerebral embolus with right-sided paralysis; this was still present to a slight degree.

On admission to hospital she was found to be 34 weeks pregnant. She had moderate dyspnœa and slight œdema of the extremities. The urine was clear. Auricular fibrillation was present; her pulse rate was 90 to 100 per minute. The apex beat was 10 centimetres (four inches) from the mid-line. A pronounced apical diastolic thrill was present. She was treated by rest in bed and by the administration of 1·2 cubic centimetres (20 minims) of digitalis three times a day at first. The dosage was gradually reduced. There had been some difficulty in controlling the heart rate, which recently had fallen to 60 per minute; but at the time of the meeting it was 68. As the patient was near full term it was decided to terminate her pregnancy by Cæsarean section followed by sterilization.

Professor Allan then showed a woman, aged twenty-five years, who had one child. She had had several attacks of rheumatic fever during the past four years, and then had been fairly well until the last menstrual period early in January. Since then orthopnœa and progressive œdema of the extremities and abdominal wall had been present.

On her admission to hospital gross cardiac enlargement with mitral stenosis was noted. Under treatment with digitalis the patient had improved considerably. The œdema was also treated with intramuscular injections of "Neptal", and much of it had disappeared. Professor Allan remarked that she was rather obese, and there was some doubt as to whether she was pregnant or whether the amenorrhœa was due to an endocrine imbalance. An Aschheim-Zondek test was to be performed. If she was pregnant he proposed to terminate the pregnancy, but to postpone for the present at least the question of sterilization, which had been recommended after consultation with a cardiologist.

Professor Allan also presented a detailed analysis of 35 cardiac cases occurring at the Women's Hospital during 1937 and 1938. There were six maternal deaths in the series. Nineteen cases were grouped in Class I and 16 in Class II. There were no maternal deaths in the first series and six in the group associated with definite signs of heart failure. Two of the deaths occurred some months after delivery. The methods of delivery employed were the following: normal delivery, 21 cases; abortion, 7; Cæsarean section, 6. One patient was undelivered. The types of anæsthesia used were the following: ether, 11 cases; ethylene and oxygen, 3; nitrous oxide and oxygen, 2; local, 2; chloroform, 1. Graphs illustrating the relationship of cardiac decompensation to parity, age and the period of pregnancy were also presented.

New Biochemical Apparatus.

DR. VERA KRIEGER conducted an inspection of the new apparatus in the biochemical laboratories. This included the following: (i) a thermostatically controlled water bath; (ii) an electrical incubator and oven with dead air space and then an outer rock silica packing as insulator; (iii) a glass blast pump; (iv) an electric hot-plate designed to boil simultaneously several series of samples in which the blood sugar was being estimated by MacLean's method; (v) an adjustable Kjeldahl distillation apparatus; (vi) the latest model of international centrifuge; (vii) a "Pyrex" apparatus for twice distilling water for the preparation of solutions for intravenous injection.

Biochemical Tests.

Dr. Krieger then demonstrated the Patterson spot test for the rapid detection of high concentrations of urea in blood, and a simple quantitative method developed in the laboratory at the Women's Hospital. She said that the details of the method had been described in THE MEDICAL JOURNAL OF AUSTRALIA of September 14, 1935, at page 340.

DR. KRIEGER and MR. T. KILVINGTON then demonstrated the preparation of extract of urine containing the antidiuretic hormone of the posterior pituitary gland. The urine was dialysed in a "Cellophane" bag till free from sodium chloride and urea, which after concentration would act as diuretics. The solution was then concentrated at 70° C. by distillation *in vacuo*. The residue was taken up in sterile distilled water, centrifuged, neutralized and used for injection.

Dr. Krieger and Mr. Kilvington then described the burn method of biological assay of the hormone in the following terms. Each of four rats was given a volume

of water by catheter into the stomach and then each received an injection of the extract into a vein in the tail. The total urine excreted was measured at regular intervals for 300 minutes. A concurrent experiment was performed on a second batch of four rats; each received the same volume of water into the stomach, but an injection of normal sterile saline solution was given into a vein in the tail. On the next day the procedure was reversed in the two groups.

Characteristic graphs of results obtained were exhibited. The first was based on the injection of urine extracts from normal non-pregnant women. No antidiuretic effect had been observed in the series. The second was based on the injection of urine extracts from normal pregnant women. Two types of curve had been obtained, (*a*) showing no antidiuretic effect and (*b*) showing a definite antidiuretic effect. The third graph represented the results of injection of urine extracts from eclamptic and pre-eclamptic pregnant women. In some cases very pronounced antidiuretic effects had been obtained.

An Unusual Condition of the Vulva.

DR. W. G. CUSCADEN presented a patient, aged seventy-two years, with an unusual growth on the vulva. She had been admitted to the Women's Hospital with an indefinite history of having had the growth for at least five or six years. The growth was associated with some tenderness, and a severe hæmorrhage had occurred two weeks before her admission to hospital. Her past history was clear and there were no other relevant symptoms.

On examination a raised, hard, nodular, red growth was found, involving the outer and inner sides of both *labia majora* and *minora*. The growth did not seem to infiltrate under the surrounding skin, was not friable, did not bleed and was not tender. Close examination proved that it had not the typical characteristics of an epithelioma. There were palpable enlarged lymph glands. Section of the growth revealed intense epithelial irritation and overgrowth; the nuclei did not look active, there were some mitotic figures, and there was no infiltration of deep tissue. There was no reaction to the Wassermann test. The growth was thought to be an atypical chronic inflammatory condition. Dr. Cuscaden said that further investigation was still in progress.

Bilateral Carcinoma of the Breast.

Dr. Cuscaden also showed a female patient, aged forty-three years, who had been admitted to hospital complaining of lumps in both breasts. For the last six years she had complained of soreness in the breasts and had noticed lumps for the last five years. Six months earlier she had begun to feel tired and had some vaginal hæmorrhages. There were no other relevant symptoms. On examination the breasts were seen to be small and to contain fairly soft nodular masses. Both nipples were retracted. The lumps were not adherent to the skin or deep tissues. Enlarged glands were present in both axillæ, in the supraclavicular and post-cervical regions. Abdominal examination revealed a hard mass filling most of the lower part of the abdomen and not movable. Vaginal examination revealed a hard, nodular mass filling almost the whole of the pelvis. Biopsy of a supraclavicular gland revealed carcinoma. The patient's blood picture was normal and the Wassermann test elicited no reaction. X ray examination of the pelvis revealed an osteoplastic type of metastasis throughout the pelvis. There were no pulmonary or mediastinal metastases. The patient's general health was very good. The pelvic mass became smaller, symptoms of pressure lessened, and the gland in the neck suddenly retrogressed. She was considered to be suffering from scirrhous carcinoma of both breasts, with bone, ovarian and lymph gland metastases. The metastases appeared to be suddenly retrogressing.

Klippel-Feil Syndrome.

DR. KATE CAMPBELL showed a new-born infant with the Klippel-Feil syndrome, characterized clinically by an extremely short neck, so that the head appeared to sit on the chest. The hair line posteriorly appeared to end at the upper limit of the thorax. Both scapulæ were elevated. X ray examination of the cervical vertebræ showed some of the vertebral bodies to be wedge-shaped. The cervical *spina bifida* found in this condition was also shown radiologically. No cervical rib was visible. The infant, though small, was well nourished and gaining weight.

Sacral Spina Bifida.

Dr. Campbell next showed an infant, aged three weeks, with a sacral *spina bifida* of meningo-myelocele type. This was associated with *talipes calcaneo-valgus* of both feet and an early hydrocephalus. The circumference of the head was increasing at an abnormal rate. X ray examination of the skull revealed digital markings of the cranial bones associated with increased intracranial tension.

Icterus Gravis Neonatorum.

Dr. Campbell then showed two infants, one aged eighteen months and one aged three months, both of whom had suffered from *icterus gravis neonatorum*. The mother of the infant aged eighteen months had a typical history of fœtal erythroblastosis. She had had three normal infants, then two full-time infants, both of whom died, one in the first week and one in the second week, from *icterus gravis neonatorum*. Then the patient was born; he had developed severe *icterus gravis*, from which he had recovered after two blood transfusions. Up to the time of the meeting he appeared quite normal in his development, and had shown no evidence of any kern-icterus or nuclear jaundice. When the patient was aged one year and five months the mother gave birth to a full-time stillborn infant exhibiting the severest form of fœtal erythroblastosis, namely, congenital œdema of the fœtus.

The second infant shown was the third child of the family. The other two children were normal. This infant had had severe *icterus gravis* and had been treated with four small blood transfusions and the administration of "Campolon". The child had recovered from the *icterus gravis* and had then developed nasal diphtheria, from which it had recovered. At the time of the meeting the child appeared healthy, and was receiving 0·3 gramme (five grains) of iron and ammonium citrate three times a day. There was no evidence of kernicterus at the time of the meeting.

Dr. Campbell went on to say that *icterus gravis* was characterized by the rapid onset of jaundice, always within thirty-six hours, generally within twenty-four hours; by the rapid increase in the jaundice; by the bile in the urine and the rise of temperature; frequently by œdema, a toxic state of the infant, enlargement of the liver and spleen, and excess of nucleated erythrocytes in the blood film. The response to the Van den Bergh test was indirect. The infant was given a blood transfusion whenever the erythrocyte count fell below three million per cubic millimetre. Better results were obtained if the amount of blood transfused was kept small; six to eight cubic centimetres of blood per pound of body weight were sufficient. Two cubic centimetres of "Campolon" given bi-weekly also appeared to be helpful. When the baby recovered a guarded prognosis was necessary. It was possible that the toxic effects of the bile on the central nervous system would produce kernicterus or nuclear jaundice, which would manifest itself in such conditions as *petit mal*, *grand mal*, spasticity, mental deficiency and athetoid and choreiform movements.

Paralysis after Nasal Diphtheria.

Dr. Campbell also showed a premature infant who had weighed 2·3 kilograms (five pounds) at birth and had developed nasal diphtheria. About four weeks later paralysis of the abdominal muscles of the right side and mild pharyngeal paralysis were present. Dr. Campbell said that the paralysis of the abdominal muscles was disappearing and that the pharyngeal paralysis had disappeared. The infant was well again and gaining in weight.

Prematurity.

Dr. Campbell then showed a premature infant, stated to be of twenty weeks' gestation. The baby in its early weeks had had an abscess on the thigh, due to hæmolytic streptococci, and the umbilicus had become similarly infected. Great difficulty was experienced in bringing about healing of the rapidly spreading and inactive umbilical septic ulcer. Treatment with "Proseptasine" was unsuccessful and produced a severe anæmia; the hæmoglobin value fell to 33%. After two blood transfusions the ulcer healed. Dr. Campbell said that the infant was ten months old and appeared to be quite normal mentally and physically. He was well nourished. He had suffered from coryza, which had been followed by otorrhœa and bronchitis.

Atresia of the Cervix Uteri.

Dr. W. D. SALTAU showed two patients with atresia of the cervix, who had been admitted to the Women's Hospital within a period of a few days. The first, a *primipara*, aged thirty-six years, at full term, was admitted to hospital on March 6, 1939, with a history of indefinite pains of twelve hours' duration. Abdominal palpation showed that the fœtus was in the left occipito-anterior position, and that the head had descended well into the brim of the pelvis.

On vaginal examination no cervix could be felt; the fornices were distended by the head, which was almost in the mid-pelvis. The pains at this stage were not very strong and were occurring at intervals of about three-quarters of an hour. The patient was placed under observation until the next morning. She had a few pains during the night; but early in the morning the pains began to occur every ten minutes and became stronger. At 11.30 a.m. she had been examined. There was still no cervical orifice to be felt. The patient was placed in the lithotomy position and, with the aid of a bivalve speculum, the vaginal vault was inspected. A small yellowish area was seen in the centre of the bulging vault, and in the centre of this was a minute opening through which a probe was passed. This was apparently the remnant of the cervical canal; and this supposition was confirmed a few minutes later, when there was a sudden small gush of *liquor amnii* followed by protrusion of a small wisp of hair of the fœtus. An attempt at dilatation of this opening or incision was considered impossible, and Cæsarean section was decided on. As the membranes had just ruptured, the classical type of operation was carried out and a living female child, weighing 3·5 kilograms (seven pounds eleven ounces) was delivered.

The patient's puerperium was complicated by sapræmia, doubtless due to deficient drainage through the minute opening. After a few days her temperature fell and remained normal, and she was transferred to the aftercare hospital on March 28, 1939. Three days later she was readmitted to the Women's Hospital with a temperature of 39·5° C. (103° F.) and a profuse foul-smelling vaginal discharge. Glycerine was injected into the uterus by means of a catheter inserted through the small cervical opening. Two days later, under a general anæsthetic, the opening was dilated with a Hegar dilator and 15 cubic centimetres of glycerine were injected. The temperature subsided in a few days, on April 9, 1939, and the patient was discharged from hospital quite well. Dr. Saltau said that the cause of the cervical atresia remained obscure. There was no history of operative treatment or of local medication to the cervix, and her previous menstrual history had been quite normal.

Dr. Saltau's second patient was also a *primipara*, aged twenty-five years, at full term, who had been admitted to hospital on March 15, 1939. On abdominal palpation the fœtus was found to be lying in the right occipito-anterior position and the head was fixed in the brim. Strong contractions had been occurring for four hours prior to the patient's admission to hospital. On vaginal examination a small transverse depression could be felt in the vault of the vagina, which was distended by the head. The patient was placed in the lithotomy position, and no normal cervix could be seen. This was replaced by a small transverse slit through which *liquor amnii* was escaping. The patient had stated that "water had been coming away for three days". Her temperature was 37·7° C. (99·8° F.). As the contractions were strong it was considered that there was no possibility that dilatation would take place; and by reason of the long interval since the membranes had ruptured it was decided that the lower segment type of operation for Cæsarean section should be performed. Some difficulty was experienced in the extraction of the child's head with forceps, as the maximum diameter had passed through the brim of the pelvis. The patient was safely delivered of a living female child weighing 3·4 kilograms (seven pounds six ounces). The retained *liquor amnii* was offensive in odour, and in view of the patient's temperature prior to operation 10 cubic centimetres of "Soluseptasine" were given intramuscularly every four hours for the first twenty-four hours, as well as 20,000 units of gas gangrene antiserum. Except for a rise of temperature to 38·4° C. (101° F.) on the evening of the second day the puerperium was uneventful.

Dr. Saltau said that in contrast with the first patient this patient had given a history of local treatment for a Neisserian infection two years previously. Dr. Saltau was doubtful whether this had played any part in the causation of the condition. Dr. Saltau had had previous experience of five cases of stenosis of the cervix necessitating Cæsarean section, but these had all followed on previous operative procedures on the cervix.

Puerperal Sepsis.

Dr. N. LENNOX SPEIRS showed a female, aged twenty-eight years, who had been admitted to the Women's Hospital on August 19, 1938, with a temperature of 40·3° C. (104° F.) and a pulse rate of 134 per minute. On August 1, 1938, she had had a normal full-time delivery of an infant. The perineum had been lacerated and sutures had been necessary. A mild *post partum* hæmorrhage had occurred. The patient had felt very well until August 9, when she had a sudden elevation of temperature and became very ill. At the time of her admission to hospital her hæmoglobin value was 75% and she was suffering from frequent rigors. Right femoral thrombosis was present.

Treatment with "Soluseptasine" and "Proseptasine" was undertaken. On August 20 culture from the blood resulted in a growth of aerobic and anaerobic non-hæmolytic streptococci. Aerobic streptococci were grown on culture from a vaginal swab. A throat swab was taken and *Staphylococcus aureus* was grown on culture, but no hæmolytic streptococci. On August 23 a course of treatment with "Uleron" was begun, two tablets being given three times a day for six days. On August 24 the hæmoglobin value was 68%; attempts at blood culture were without result. On August 25 a transfusion of 568 cubic centimetres (20 ounces) of blood was given. On August 29 the hæmoglobin value was 72%; another transfusion of 568 cubic centimetres (20 ounces) of blood was given. On August 31 the patient was feeling very well, but her temperature was still elevated. Her progress continued. On September 10 her temperature was normal. The patient felt very well, but she was kept in hospital until the femoral thrombosis had cleared up. On October 4 she was discharged.

On October 8 the patient was readmitted to hospital, complaining of pain in the left side of the neck and the left shoulder. She felt ill. Her temperature was 38·5° C. (101·2° F.) and her pulse rate was 112 per minute. Radiological examination revealed a destructive lesion of the upper and anterior aspect of the fourth cervical vertebra. On October 14 her temperature had fallen. On October 18 she was fitted with a plaster collar to keep her head in extension. On October 21 she was allowed to sit out of bed. Her temperature rose to 38·7° C. (101·6° F.). She complained of a tingling sensation in the arms and lost the power of her left arm. She was sent back to bed. On October 22 her head was immobilized more effectively. Her left arm was placed in an abduction splint. On

November 9 her left arm was strong again, and on November 20 the patient was once more allowed to sit out of bed. On December 12 radiological examination revealed further destruction about the upper margin of the fourth cervical vertebra. On December 24 the patient was discharged, still wearing the plaster splint and collar.

Post-Abortal Bacillus Welchii Infection.

Dr. Speirs also showed a patient, aged thirty years, who had three children. She had had an abortion three years earlier. She had been admitted to the Women's Hospital at 1 a.m. on March 3, 1939, complaining of lower abdominal pain, vomiting, headache, shivering and slight discharge *per vaginam*, of a few hours' duration. She had been two months pregnant, and an attempt to procure an abortion had been made four days before her admission to hospital. She had remained well since then until the onset of the symptoms of which she was complaining.

On her admission to hospital her temperature was 38° C. (100·4° F.) and her pulse rate was 118 per minute. Vaginal examination revealed that the fundus was anteverted and was the size of an eight weeks' pregnancy. It was not tender. The os was patulous and the fornices were clear. At 10 a.m. on the same day the patient was cyanosed and slightly jaundiced. Culture from the blood resulted in a growth of *Bacillus welchii*, and culture from the urine yielded *Bacillus welchii* and a few non-hæmolytic streptococci. *Bacillus welchii* and *Bacillus coli communis* were grown on culture from vaginal swabbings. The hæmoglobin value was 90% and the urine was blood-stained. Gas gangrene antiserum was given, and saline solution was administered by the continuous intravenous drip method.

At 5 p.m. on the same day Dr. Speirs performed a total hysterectomy. The uterus was purplish in colour and contained a foul-smelling necrotic placenta. There was some free fluid in the abdomen. "Soluseptasine" was given and the continuous rectal administration of water and sodium bicarbonate was begun. Alkaline drinks were given by mouth. Morphine was given and dextrose was injected intravenously. From March 8 to March 27 inclusive, 910,000 units of gas gangrene antiserum were given; 710,000 units were given intramuscularly and 200,000 intravenously.

On March 11 half of the patient's urine consisted of pus and blood. On March 14 her urine output was about one ounce per day, two-thirds consisting of pus. She had a cough, with foul-smelling necrotic sputum, and twitchings of the face. Her mental condition was poor, and generalized œdema was present. On March 17 the patient's urinary output began to improve. On March 22 it had reached 2·3 litres (four pints) and the œdema had gone. On this day the blood urea content was 289 milligrammes. *Bacillus coli communis* alone was grown on culture from the urine. On March 28 the use of gas gangrene antiserum was discontinued. The hæmoglobin value was estimated at 35% on April 7, and on April 8 a transfusion of 568 cubic centimetres (20 ounces) of blood was given. On April 15 the blood urea content was estimated at 84 milligrammes. A urea concentration test was performed on this date, and the urea concentrations at the end of the first, second and third hours were 0·51%, 0·76% and 0·80% respectively. The total amount of urea excreted during the three hours was 2·3 grammes.

A MEETING of the New South Wales Branch of the British Medical Association was held on April 20, 1939, at the Royal Alexandra Hospital for Children, Sydney. The meeting took the form of a number of clinical demonstrations by members of the honorary staff of the hospital.

Toxic Anæmia.

DR. L. A. DEY showed a boy, aged five and a half years, who had been admitted to the hospital on March 21, 1939. He had previously had pertussis. Vomiting and diarrhœa with abdominal pain had been present for one month prior to his admission to hospital. He had had a cough for two weeks; it was growing worse and the child was feverish.

On examination the child was seen to be very pale. His temperature was 38·9° C. (102° F.) and his pulse rate 176 per minute. The respirations numbered 36 per minute. Signs of bronchopneumonia were detected in the chest. No abnormality was found in the other systems.

A blood count gave the following information. The erythrocytes numbered 1,750,000 per cubic millimetre and the hæmoglobin value was 27%; the leucocytes numbered 3,600 per cubic millimetre; 37% were polymorphonuclear cells, 54% were lymphocytes, 1% were monocytes, 4% were lymphoblasts and 2% were smear cells. The platelets numbered 58,400 per cubic millimetre.

Radiological examination of the chest revealed pronounced chronic bronchitis with bilateral bronchiectasis. Radiological examination of the skeleton gave the following information. The bones of the inferior extremities appeared denser than normal and somewhat widened, especially the lower ends of the femora and to a less degree the tibia and the first metatarsals. The changes were unusual and not typical of leuchæmia. There was no evidence of any periosteal change and the skull appeared normal. No gross changes were present in the ribs or upper extremities.

Examination of the urine revealed the presence of urobilinogen.

On March 22 a blood transfusion was given and evoked a slight response. On March 28 a further transfusion increased the number of erythrocytes to 2,250,000 per cubic millimetre and caused a fall in the number of immature cells. There had been a steady improvement since; at the time of the meeting the erythrocytes numbered 3,750,000 per cubic millimetre and the hæmoglobin value was 37%. The leucocytes numbered 8,000 per cubic millimetre, and there were no immature cells. The child's chest was clear and his general condition was good.

Chronic Malaria and Severe Anæmia.

Dr. Dey next showed a female child, a native of Samoa, aged two years and eight months, who had been admitted to hospital on April 9, 1939. She had been in Sydney for two weeks and was on her way back from Papua to Samoa. On the day before the onset of her illness she had been given quinine. At the time of her admission to hospital she had been ill for four hours; she had been feverish, and had vomited and passed blood in the urine. She had previously had malaria and hæmaturia.

On examination the child was seen to be very pale. Her temperature was 38·4° C. (101° F.) and her pulse rate was 166 per minute. Splenomegaly, slight icterus of the conjunctiva and hæmaturia were present.

No parasites were seen in a blood smear. A blood count revealed microcytic hypochromic anæmia, monocytosis and thrombocytopenia. The erythrocytes numbered 1,250,000 per cubic millimetre and the hæmoglobin value was 18%. The colour index was 0·74. The leucocytes numbered 9,300 per cubic millimetre; 44% were polymorphonuclear cells, 40% were lymphocytes, 15% were monocytes and 1% were eosinophile cells. Platelets numbered 100,000 per cubic millimetre. The erythrocytes were of normal fragility.

Blood was present in the first ward specimen of urine, but none was found in later specimens on microscopic or spectroscopic examination.

A blood transfusion was given immediately and evoked a pronounced response. In four days the erythrocytes were almost doubled in number and the hæmoglobin value was much increased. Dr. Dey said that in view of the history of hæmaturia and the absence of parasites from the blood no quinine had been given up to the time of the meeting.

A Case for Diagnosis.

Dr. Dey's third patient was a boy, aged thirteen months, an only child, who had been admitted to the hospital on April 5, 1939. The parents had noticed spots on the

child's body, face and limbs for three months. The child was always pale and the abdomen was increasing in size.

On examination the child was seen to be pale and flabby and mildly feverish. Widespread purpuric spots and bruises were evident, and pronounced hepatomegaly and splenomegaly were present. A mass was palpable in the region of the left kidney, behind the spleen. No enlarged glands were present.

An X ray examination was made of the chest, skull and long bones, and gave the following information. There was no evidence of neuroblastoma, except for some doubtful periosteal reaction along the long bones, which the radiologist thought might have been due to one of the blood dyscrasias and possibly neuroblastoma. The changes were only doubtfully abnormal.

A blood count on April 7 gave the following information. The erythrocytes numbered 2,930,000 per cubic millimetre, the hæmoglobin content was 297 grammes per 100 cubic centimetres, the colour index was 0·7, and the corpuscle index was 58. Reticulocytes numbered 5·6%, and a megaloblast and two normoblasts were seen. The leucoytes numbered 9,600 per cubic millimetre; 23% were polymorphonuclear cells, 61% were lymphocytes, 6% were monocytes, 3% were eosinophile cells and 2% were myelocytes. Four extremely immature blast cells were seen, two of which suggested lymphoblasts. Platelets numbered 20,000 per cubic millimetre.

The child's condition was diagnosed as severe anæmia, microcytic and hypochromic in type. Neutrophile leucopenia and lymphocytosis were present, and immature cells of all series were present in the circulating blood.

On April 16, after a transfusion of blood, a further blood count was made. The number of erythrocytes was almost unchanged and there was no evidence of response on the part of the bone marrow. Neutrophile leucopenia was still present. Lymphocytes were present in normal numbers, but some immature forms were observed. There was a pronounced diminution in the number of platelets. Dr. Dey said that the diagnosis appeared to lie between neuroblastoma of the left kidney with metastases and an atypical leuchæmia, though the tumour in the kidney region and the age of the child were against the latter diagnosis.

Chronic Colitis.

Dr. Dey next showed a female child, aged eight years, who had been admitted to hospital on October 24, 1938. The mother stated that the child had been bleeding from the bowel off and on for several weeks prior to her admission to hospital. The bleeding usually occurred with the motions, but it occasionally occurred at other times. The child had lost a considerable quantity of blood and the hæmorrhage was gradually increasing. She had had no diarrhœa before her admission to hospital.

At the time of her admission to hospital the child also had an attack of bronchitis. During this period there was much bleeding *per rectum* and she was given a blood transfusion. The chest disorder and the bleeding still continued and another blood transfusion was given. The bronchitis finally cleared up, and the child was examined *per rectum* under anæsthesia. No bleeding and no polypus was seen. She was having four to six motions per day.

On November 24, 1938, an X ray examination was made. The radiologist reported that the appearance of the bowel suggested pronounced mucous colitis involving the whole of the colon.

Several pathological tests were carried out. *Bacillus proteus* was grown on culture. There was no response to the Mantoux test. The fæces were found to contain no ova or parasites.

The child continued to have several motions per day, the fæces containing blood. She was given a starch and opium enema daily, and was fed on an apple diet for some time. At the time of the meeting her temperature was normal and there was no blood in the stools. She was having one or two motions per day. Dr. Dey said that the child had had five blood transfusions. After a long period of diarrhœa with much wasting and secondary anæmia she was at last gaining weight.

Tetanus.

Dr. Dey finally showed a boy, aged eleven years, who had been admitted to the hospital on March 26, 1939, with the history that he had run a nail into his foot ten days previously. On the night before his admission to hospital he had complained of stiffness of the jaws and severe pains in the back.

On examination the boy appeared pale. There were small black spots on the sole of the left foot, which were thought to be the perforating wound. The appearance of the face was characteristic; the brow was furrowed and the eyebrows were elevated, and *risus sardonicus* and pronounced trismus were present. The temperature was 36·9° C. (98·4° F.) and the pulse rate was 104 per minute. The back and neck were rigid and arched, and the tone of the abdominal muscles was increased. There was no spasticity of the limbs. All the reflexes were exaggerated.

On his admission to hospital the child was given 30,000 units of tetanus antiserum by intravenous injection and 50,000 units by intramuscular injection, and "Sodium amytal" by mouth. He had two spasms a few hours later, and paraldehyde was given by the nasal tube. The next day he was restless, and paraldehyde was given every four hours. A further intramuscular injection of 30,000 units and a further intravenous injection of 30,000 units of tetanus antiserum were given. On the third day he had a minor spasm affecting the face only. The four-hourly administration of sedatives was continued, and a further intramuscular injection of 60,000 units of tetanus antiserum was given. On the fourth day he had major spasms. Another intramuscular injection of 50,000 units of tetanus antiserum was given. He had minor spasms for the next four days, during which time 30,000 units of tetanus antiserum were given. Serum treatment was suspended for a few days; but the patient again became restless and his stiffness was still severe, so treatment was recommenced.

On the fifteenth day the wound was excised and serum treatment was continued until the eighteenth day and then suspended. Dr. Dey said that since the excision of the wound the patient had been progressing steadily, and on the twenty-fourth day, the day of the meeting, he was able to feed himself and had lost much of his stiffness. He had received a total of 530,000 units of tetanus antiserum, of which 60,000 units were given intravenously and 330,000 were given in the first week. Dr. Dey remarked that the interest in this case lay in the exacerbation of the symptoms in the period without serum treatment and the child's immediate improvement after excision of the wound.

Rheumatic Carditis.

DR. LAURENCE HUGHES showed a female, aged seven years, who had been first admitted to the hospital at the age of four and a half years. At that time the apex beat was visible over two intercostal spaces, and a blowing systolic mitral murmur was present. The child had no tonsils or adenoids. She had had a further attack four months later, when blowing systolic and diastolic murmurs were present at the mitral area and axilla. The third attack had occurred one year prior to the meeting. The apex beat was just outside the nipple line, and a pronounced blowing systolic murmur was heard at the mitral area and conducted into the axilla. The aortic and pulmonary areas were clear. During this attack the child had been very ill, cyanosed and dyspnœic, and the sedimentation rate had been high. Radiological examination had suggested enlargement of the aortic bulb, possibly aortic stenosis. At the time of the meeting the child's condition was good and the sedimentation rate was normal.

Dr. Hughes's second patient was a boy, aged twelve years, who had had his first attack of rheumatism when six years of age. He had had a second attack in December, 1937. The apex beat was diffuse, and a mitral systolic and diastolic murmur was present. The child had no tonsils or adenoids. He had had an attack of chorea in June, 1938, and a further attack in September, 1938. At the time of the meeting the apex beat was now in the sixth interspace in the anterior axillary line. There was a

mitral systolic murmur conducted into the axilla. A rough systolic and diastolic murmur was audible at the aortic area. The child's general condition was good and the condition of the heart was unchanged. The sedimentation rate was normal.

Dr. Hughes next showed a girl, aged nine years, who had been first admitted to hospital in June, 1937, with a history of having had scarlet fever previously. She had had a rapid pulse and had been breathless on exertion for several months. The apex beat was outside the nipple line, and presystolic and systolic mitral murmurs were audible. Tonsillectomy was performed on July 10, 1937. The child had been readmitted to hospital in December, 1938, with the history that she had had a convulsion followed by unconsciousness for several hours, twenty-four hours before her admission. A mitral presystolic murmur was present. The sedimentation rate was normal. Dr. Hughes said that her general condition was fair and the sedimentation rate was normal when last tested.

Dr. Hughes's fourth patient was a girl, aged ten years, who had first become ill four years earlier, when she developed chorea. She had a similar attack eighteen months later. At that time a systolic murmur was present at the mitral area. The apex beat was in the fifth intercostal space, just outside the mid-clavicular line. Later on she developed acute rheumatism and had a further attack of chorea some six months later. A systolic murmur was still present at the mitral area. The aortic area was clear. During this attack the child had developed a presystolic mitral murmur.

The present attack had commenced two weeks prior to her admission to hospital in November, 1938. She had pains in the legs and breathlessness on exertion.

On examination the child's pulse was found to be regular and forcible. The systolic blood pressure was 120 and the diastolic pressure was 60 millimetres of mercury. Diffuse pulsation was present in the fourth, fifth and sixth intercostal spaces. The apex beat was in the sixth intercostal space, 11·3 centimetres (four and a half inches) from the middle line. A loud blowing systolic murmur was heard at the mitral area, propagated into the axilla. There was a soft blowing to-and-fro murmur at the aortic area. No œdema was present. The tonsils were somewhat enlarged, and the liver was just palpable below the costal margin. The sedimentation rate at this time was 75 millimetres after two hours. Dr. Hughes said that the child had had periods of improvement and relapse since then. At the time of the meeting the sedimentation rate was 30 millimetres after two hours, and her general condition was fair.

Post-Encephalitic Syndrome.

Dr. Hughes finally showed a girl, aged three and a half years, who had been admitted to hospital on March 13, 1939. She had had a cold for one week, and on the day of her admission to hospital she had a convulsive seizure which lasted for half an hour. She continued to twitch for an hour. There was no history of dietetic indiscretion or of previous convulsions. The child's mentality was normal.

On examination generalized convulsions were present, and both arms and legs were twitching. There was no neck rigidity, spine sign or Kernig's sign. The child continued to have convulsions, and lumbar puncture was performed.

On March 18, 1939, rigidity of the spine and neck developed. The head and eyes were turned to the left. By March 23, 1939, the upper limbs were spastic. Since then the child had much improved. The spasticity and rigidity had disappeared. She was mentally altered and laughed easily and vacantly. She was unable to sit up and was unintelligent.

Several pathological tests were made. The cerebro-spinal fluid was clear and contained no cells. The total protein content was 40 milligrammes per 100 cubic centimetres. The total chlorides were 760 milligrammes *per centum*. Glucose was present. No globulin was present and the fluid was sterile. Radiological examination of the skull revealed no abnormality, and the fundi were normal.

Cœliac Disease.

DR. R. A. R. GREEN showed a boy, aged two years, who had been suffering from diarrhœa and vomiting and had been losing weight for four months. The fæces were pale and offensive, and of enormous bulk. The child's abdomen was becoming enlarged.

On examination the child appeared pale and sallow. The abdomen was distended, the buttocks and thighs were wasted, and there was generalized hypotonia.

Radiological examination of the long bones revealed unusual transradiancy. A blood count gave the following information. The erythrocytes numbered 4,630,000 per cubic millimetre, the hæmoglobin value was 60%, and the colour index was 0·65. The Mantoux test elicited no reaction. Analysis of the stools showed that the total fat content was 51·6%, of which 84·2% was split fat and 15·8% unsplit fat. A sugar tolerance test was performed; there were 94 milligrammes per 100 cubic centimetres after one hour and 82·5 milligrammes per 100 cubic centimetres after one and a half hours.

Lead Poisoning.

DR. C. L. S. MACINTOSH showed a boy, aged two years and ten months, who had been admitted to hospital on February 21, 1939. There was nothing of note in the previous history, beyond two attacks of bronchopneumonia.

The child had been retching and vomiting since an attack of "gastric influenza" in October, 1938. The vomiting occurred either before or after breakfast, generally after lunch, and not as a rule at tea time. The vomiting was accompanied by retching that might be sufficiently severe to cause exhaustion. He had lost weight and was irritable, frequently waking up at night crying. He was drowsy during the day and he had always been pale. His mother was receiving treatment for anæmia; the possibility of lead poisoning in her case had been excluded. The boy was not constipated. It was believed that he had swallowed some small toy some time ago.

On examination the child appeared pale, fairly well nourished and not very intelligent. Clinical examination otherwise revealed no abnormality.

For the first ten days after his admission to hospital the retching and vomiting were very troublesome. Vomiting was of an extremely projectile type, and the vomitus consisted of a greenish-yellow fluid. This had gradually subsided. Three weeks prior to the meeting a decided double foot-drop was noticed. The child had made slow, steady progress since his admission to hospital. His colour had improved, he was less irritable, and his mental development was not striking.

Several investigations were undertaken. The optic fundi were normal; the disks were paler than normal and there was no swelling. Radiological examination of the lungs and alimentary tract revealed no abnormality and no evidence of any opaque body. The radiologist reported increased density at the ends of the long bones and ribs, and that the appearances were very suggestive of lead poisoning. Neither the Wassermann nor the Mantoux test elicited any reaction. The child's blood was found to belong to Group II. Several blood examinations were made, and the average results were the following. The erythrocytes numbered 2,710,000 to 3,990,000 per cubic millimetre. The hæmoglobin value was 45% to 48%, and the colour index 0·73 to 0·85. The corpuscle index was 54 to 79. There was a central pallor; the cells were mainly of the microcytic type and of regular shape. There was a coarse stippling of the cells, and of these cells there were between 100,000 and 69,000 per cubic millimetre of blood. An occasional normoblast was detected. Reticulocytes numbered about 2·2%. The number of leucocytes had varied between 13,000 and 7,200. The lymphocytes were increased in number. An average count was the following: polymorphonuclear cells, 47%; lymphocytes, 43%; monocytes, 6%; eosinophile cells, 4%. There were about 500,000 platelets per cubic millimetre. The findings indicated a pronounced anæmia of the hypochromic and microcytic type with lymphocytosis and an increase in the number of stippled cells.

The urine was acid and free from albumin and sugar on many examinations. The deposit consisted of large phosphate crystals. The lead content was 0·3 gramme.

Dr. Macintosh said that the child resided in an old dwelling that had had many superadded coats of paint in the past, the last being long ago. The many coats of paint were flaking, and it had been the child's custom to pick these flakes off the walls and eat them. The child had made good progress; he was treated with di-sodium phosphate, 0·3 gramme (five grains) three times a day.

(To be continued.)

Obituary.

WALTER CHARLES MANSFIELD.

WE are indebted to Dr. Robert Godsall for the following appreciation of the late Dr. Walter Charles Mansfield.

With the passing of Walter Charles Mansfield his many patients have lost a trusted friend and consultant.

Educated first at Hayfield School and later at Sydney Grammar school, he began his medical course in 1899. He took excellent microphotographs of biological sections, which he distributed to his fellow students and which helped us greatly to understand the mysteries of that, to us, unknown science.

In the years following his graduation in 1905 I met Mansfield in England, Scotland and various Continental clinics, and saw the thoroughness with which he was preparing for his future specialty.

Appointed to the Royal Prince Alfred Hospital in 1911, we were colleagues for eighteen years, and no one could have found a more loyal and willing friend. Possessed of a high degree of surgical ability, he was always ready to help in our difficulties, rejoicing in our successes and sympathizing in our failures.

On establishment of the Royal Australasian College of Surgeons he was elected a foundation fellow. A lucid teacher, he became lecturer in diseases of the ear, nose and throat until his resignation in 1929. About this time his health began to give his friends serious concern and he felt he could no longer cope with the strenuous work of a large public hospital, and it was with sincere regret we learnt of his decision to retire. He still carried on his private work, and during the last few years his courage in the face of increasing physical disability called forth our warmest admiration.

An incorrigible storyteller, Mansfield had a tale to suit all palates and all occasions; he met the world with a smile and the world in turn smiled with him. His manner of passing truly exemplified the man—a quiet courage in the face of intense physical discomfort will always remain an example and a precious memory to his many friends and patients.

WILFRED NORMAN DAVIES.

WE regret to announce the death of Dr. Wilfred Norman Davies, which occurred on May 29, 1939, at East Malvern, Victoria.

Special Correspondence.

CANADA LETTER.

FROM OUR SPECIAL CORRESPONDENT.

THE development of health insurance plans of various kinds is becoming more and more popular in Canada. A typical instance is the Associated Medical Services Incorporated. This body was organized in Toronto in 1934 as the result of the recommendations of a committee appointed by the Ontario Medical Association on health insurance. Anyone interested in health insurance will understand that all such plans involve the collection of large quantities of data, and this was no exception. For a time the provincial association helped with the expense of inaugurating this organization, and then it was left to operate independently.

The rates are as follows. To join costs a patient $2.00 per month, $24.00 per year. The charge to a family is reduced, at the rate of twenty-five cents less for the first dependant, another twenty-five cents less for the next dependant, and so on. The physicians are paid according to the minimum rates of the tariff of charges laid down by the Ontario Medical Association. These figures are admittedly liable to alteration as time goes on, since the cost of medical care in Canada is not yet clearly defined. As a beginning, however, the rates are close enough.

The scope of the services rendered is important. The "A.S.M." offers a complete essential medical service, and there is no specialty which it does not cover. "Essential", of course, needs some definition, and in general it may be said that it requires that calls must be such as would be made by a doctor for a given patient who did not belong to the "A.S.M." Such a thing as annual health examinations, however, would not be considered as essential. The scheme allows of free choice of doctor by the patient, and no third person intervenes.

Another form of health insurance is to be found in the hospital-care insurance plan. This provides for hospitalization and various other benefits. There are several large plans of this kind in the United States, the largest being in New York, where over a million are enrolled. Within the last few months two new hospital-care insurance plans have been set up in Canada, one with its headquarters in Winnipeg, and another at Nelson, British Columbia. They vary slightly in the benefits provided, one giving semi-private accommodation and the other public ward. But the fundamental idea of insurance against hospital expenses is the same.

What will be the eventual result of such developments as these? Will they solve the problem of assuring the medical man a steady return for his work whilst enabling the majority of the population to obtain medical attention without fear or favour, fear of crippling their finances in severe illnesses, or favour of free treatment as public patients, and all that that means?

It is hard to say. Of course it may be asked, what about State medicine? Is there any likelihood of its being put into force in Canada? The best guide in this matter naturally is the attitude of the Parliament. Judging by that, we are not yet within measurable distance of State medicine as a formal measure. Recently the whole matter was aired in a long-winded discussion in the House, which was started off by a motion that we adopt the same plan of State-aided medical services as is in force in the United States.

The discussion was as free as one could desire in a democratic country; but was it effective? Not very. Except perhaps in this respect: it came out (although it should have been known already) that actually there was a great deal being done in the country along these lines, and that there had been much discussion on it before. The previous government, for example, had considered the matter and had found that they would be exceeding their powers in acting on it, since it would infringe on provincial rights. Then the organized medical profession of the country had worked out a well-planned, carefully considered scheme of health insurance ready for submission to the government. More than one province already had health insurance statutes on their books. British Columbia, for example, had even passed such a statute, but had not put it into force, partly because the conditions under which it was proposed to operate it were opposed almost unanimously by the medical profession of the province. Then, in the prairie provinces there is in force, and has been for some time, a municipal doctor system which is as close to State medicine as one could wish to get.

Apparently, however, our legislators had cared for none of these things, and only woke up to their presence collectively when the motion mentioned above was brought forward. But apart from the fact that we are not in the habit of copying other countries (although we might well do so sometimes) there was one very cold and indigestible fact which could be in no way disregarded. What would State control of medical services cost? The Minister of Health brought this fact forward in all its most disagreeable array. Apart from every other unwelcome aspect of State medicine, the cost to the country would be $250,000,000.00 *per annum*.

Well, nowadays countries do not seem to mind rivalling the Egyptians in building pyramids—of debt. The United States may easily claim undisputed precedence in this respect. But Canada just simply cannot handle a stone of that weight for her pyramid. That is why State medicine is not close by. These voluntary and local schemes I have referred to will continue to grow, and possibly there will be some coordination of them under State supervision later on, although I do not see that that will necessarily improve them.

Post-Graduate Work.

COURSE IN OBSTETRICS AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine announces that a residential post-graduate course in obstetrics will be held at the Women's Hospital, Crown Street, Sydney. The work of the course will be supervised and will be of a practical nature. Patients will be allotted to post-graduates, who will have the opportunity of following the patients right through their stay. In addition to the clinical work the following talks will be arranged.

Sunday, July 30.

10.30 a.m.: Introduction and explanation of course. Film on abdominal palpation, Dr. T. Dixon Hughes.

2.30 p.m.: "The Care and Feeding of Infants", Dr. Selwyn Harrison.

Monday, July 31.

Morning: Rounds of hospital with superintendent. "Clinical Pathology and Biochemistry as applied to Obstetrics", Dr. Beatrix Durie.

Afternoon: "Diagnosis and Treatment of Disproportion", Dr. A. J. Gibson.

Tuesday, August 1.

Morning: Indications for the application of forceps and demonstration, Dr. R. McD. Bowman.

Afternoon: Demonstration of X ray films, Dr. D. G. Maitland.

Wednesday, August 2.

Morning: Antenatal clinic.

Afternoon: "The Diagnosis and Treatment of Persistent Occipito-Posterior Presentation", Dr. H. McCredie.

Thursday, August 3.

Morning: "Toxæmia of Pregnancy", Dr. R. B. C. Stevenson.

Afternoon: "Medical Complications Associated with Pregnancy", Dr. R. Jeremy.

Friday, August 4.

Morning: "Diagnosis and Treatment of Breech Presentation", Dr. F. A. Bellingham.

Afternoon: "Treatment of Hæmorrhage in the Last Three Months of Pregnancy", Dr. T. Dixon Hughes.

Saturday, August 5.

Morning: "Résumé of Investigation, Diagnosis and Treatment of Puerperal Sepsis", Dr. J. Chesterman.

The fee for the course is five guineas, and membership is limited to six. Applications should be made as early as possible to the Secretary, New South Wales Post-Graduate Committee in Medicine, the Prince Henry Hospital, Little Bay. The fee should accompany the application.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1925 *to* 1935, of Queensland, as duly qualified medical practitioners:

Elias, Leaton, M.B., 1938 (Univ. Sydney), Gladstone.

Hertzberg, Reuben, M.B., 1939 (Univ. Sydney), Toowoomba.

Löfkovits, Leopold, M.D., 1932 (Univ. Siena), Brisbane.

Tenny, Alan Percival, M.B., B.S., 1937 (Univ. Melbourne), Toowoomba.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Speight, Richard John, M.B., 1931 (Univ. Sydney), 37, Johnston Street, Annandale.
Reid, William Lister, M.B., B.S., 1931 (Univ. Adelaide), 54, Cranbrook Road, Bellevue Hill.

The undermentioned have applied for election as members of the Victorian Branch of the British Medical Association:

Weyman, Irwin James, M.D. (Genoa), 188, Cotham Road, Kew.
Colin, Ernest, L.R.C.P. and S. (Edinburgh), Eye and Ear Hospital, East Melbourne, C.2.

Diary for the Month.

JUNE 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 20.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 21.—Western Australian Branch, B.M.A.: Branch.
JUNE 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 26.—Queensland Branch, B.M.A.: Council.
JUNE 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 28.—South Australian Branch, B.M.A.: Annual meeting.
JULY 6.—South Australian Branch, B.M.A.: Council.
JULY 27.—South Australian Branch, B.M.A.: Branch.
JUNE 28.—Victorian Branch, B.M.A.: Council.
JUNE 29.—New South Wales Branch, B.M.A.: Branch.
JULY 4.—New South Wales Branch, B.M.A.: Council (quarterly).
JULY 5.—Victorian Branch, B.M.A.: Branch.
JULY 5.—Western Australian Branch, B.M.A.: Council.
JULY 6.—South Australian Branch, B.M.A.: Council.
JULY 7.—Queensland Branch, B.M.A.: Branch.
JULY 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments.

Dr. J. H. Rickeard has been appointed Government Medical Officer at Wyong, New South Wales.

* * *

Dr. I. B. Sebire and Dr. A. E. Newton-Tabrett have been appointed Medical Officers in the Medical Branch of the Department of Public Instruction of New South Wales.

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Dr. S. Krantz has been appointed Honorary Assistant Surgeon and Dr. J. L. S. Scott has been appointed Honorary Clinical Assistant to the Surgical Section of the Adelaide Hospital, Adelaide, South Australia.

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Dr. C. R. Wiburd has been appointed Chief Medical Officer for the Northern Territory, according to the provisions of the *Hospitals Ordinance*, 1928-1932, of the Northern Territory.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages XX to XXII.

CHILLAGOE HOSPITAL, CHILLAGOE, QUEENSLAND: Medical Officer.
ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Honorary Officers.
THE ANTI-TUBERCULOSIS ASSOCIATION OF NEW SOUTH WALES: Registrar.
TARA DISTRICT HOSPITAL, TARA, QUEENSLAND: Medical Officer.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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